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THIS WORK

IS INSCRIBED TO MY PRECEPTORS,

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DISEASES OF INFANCY AND CHILDHOOD.

SECTION I.

INFANCY AND CHILDHOOD.

I. DEFINITION OF INFANCY AND CHILDHOOD.

Infancy, or the nursing age, is the period of life during which the child is at the breast. It extends from birth to the twelfth month.

Childhood is the succeeding period, extending to puberty. In addition, it is customary to divide the period of childhood into two parts—the first extending from the end of the first to the sixth or seventh year, or the beginning of the second dentition; the second, from this to puberty.

Epstein would include as **newborn** all infants up to the third month.

II. MORBIDITY.

The Newborn Infant.—The diseases of the newborn infant are, for the most part, septic in nature, and attack the infant within a short time after birth.

Conditions favor the diseases common at this time of life. The skin is not fully formed, is in process of desquamation, and bacteria easily gain access to the circulation. The umbilicus is an open wound, receptive of infection. The mucous membranes of the intestine, mouth, eye, and ear are other avenues of entrance for bacteria. There is a tendency for minor infections to become general at this period. The artificially fed infant is, in addition, exposed to the dangers which necessarily accompany the introduction into the body of a foreign food with its attendant uncleanliness, and is also deprived of the protective bodies (alexins) contained in the mother's milk. With new surroundings, in a new atmosphere, with new appliances for maintaining the body-heat (such as the clothes), and with careless handling, it is obvious that the newborn infant is peculiarly exposed to bacterial diseases.

Childhood.—If we study the statistics of any large pediatric clinic, it will at once be apparent that up to the tenth year of life those diseases which affect the respiratory apparatus form nearly two-fifths of the cases. Next in order of frequency are the diseases of the digestive tract; and, lastly, the acute infectious diseases, such as the fevers and exanthemata. Of 53,040 cases met with during five years in an ambulatory clinic, there were 20,207 cases of diseases of the respiratory organs, 17,058 of the gastro-enteric tract, and 2409 of the acute infectious diseases. If the morbidity is analyzed still further, it is seen that in the nursing period intestinal disturbances are the most frequent. The numerous flora of bacteria and their toxins in the intestine of the infant rather predispose to infections from that source. These bacteria may invade the mucous membrane of the intestine, and in certain disturbances of the functions of the gut obtain access to the circulation. The respiratory diseases become more frequent in the second year, and reach their maximum frequency between the second and third years. Constitutional diseases, such as rachitis, appear in the second half-year of life, and reach their greatest frequency during the period from the tenth to the fifteenth month. On the other hand, the acute infectious diseases, such as the exanthemata, are more common from the fifth to the eighth year. Scarlet fever, with its kidney complications, is most frequent at the fourth year (Escherich), diminishing at the ninth year. The period extending from the second to the fourth year is also notable for the frequency of the so-called "filth infections" of Feer. Children infect themselves with dirt and dust at play, at meals or in their intercourse with one another. For this reason, diphtheria as well as pertussis and tuberculosis (Escherich) attain their maximum frequency at this period.

III. MORTALITY.

The mortality of infants is large, and is highest among the poor of cities of the first magnitude. With the well-to-do, artificial feeding is resorted to for social reasons; among the poor, a mother who is forced to work is compelled to deny the breast to her child. The vast majority of deaths occur among the artificially fed infants in all countries. In England fully two-fifths of the whole number of deaths occur before the tenth year; one-fourth occurring before the termination of the first year of life. These figures, given by Williams, correspond quite closely to those of Eröss, of Germany, and to what is known to be true of America. The mode of living among the poor, and the lack of complete, or even partial, isolation in contagious diseases, tend to increase this great mortality among infants and children.

The following table was furnished to the author by the New York Board of Health, and shows at a glance the death-rate among infants and children at various ages:

Year.	Under one year.		One year to five years.		Five to ten years.		Ten to fifteen years.	
	Deaths.	Death-rate.	Deaths.	Death-rate.	Deaths.	Death-rate.	Deaths.	Death-rate.
1900 . .	16,640	191.7	9196	29.54	2073	5.86	863	2.86
1901 . .	15,467	173.6	8789	27.49	2130	5.87	904	2.92
1902 . .	15,526	164.8	8862	26.27	2065	5.39	889	2.72

The death-rate per thousand in diarrhœal disease in old New York of infants below a year of age was as follows:

In 1890	2.55
In 1891	1.71
In 1892	1.52

There has been in New York at least a gradual diminution of the death-rate in one of the principal causes of mortality among infants. This is quite easily explained by certain conditions which obtain to-day and which were absent years ago. Cows' milk, which is the principal food of artificially fed infants, is much improved as to quality, through the efforts of the authorities, as compared with the standard in vogue some years ago. The establishment, also, of milk laboratories for the distribution to the poor of pasteurized or sterilized milk in separate nursing portions, and the teaching of hygienic measures directed toward the prevention of disease, have tended vastly to improve the conditions in this social stratum of society. We hardly see to-day, what formerly was quite a common sight, a baby fed from one bottle throughout the twenty-four hours. Even the lowliest mother to-day understands the necessity of cleanliness in the preparation of the infant's food. If mothers could be taught more fully methods of prophylaxis and hygiene we could still further reduce the death-rate among infants.

IV. SUDDEN DEATH AMONG INFANTS AND CHILDREN.

In many diseases prevalent among infants and children, sudden death is the outcome. In hospital services, at least, it is not uncommon for an infant, who is apparently doing well, to be overtaken in the next few moments by sudden death. It is not surprising, therefore, that in New York alone, in the year 1902, no less than 594 infants below the age of one year have been reported to the Board of Health as having died suddenly. The total number of children dying in this manner in 1902 was 717. These infants and children are reported to the Board of Health as dying suddenly with or without convulsions. In the first year of life, therefore, sudden death is very frequent, and the statistics given by the New York Board of Health are confirmed by those of other cities of the first magnitude. Richter has recently shown that in Vienna 1797 cases of sudden death occurred during the years from 1897 to 1901 in children from the first day of life to the fifteenth year; 1525 of these deaths occurred in infants below the age of one year. In New York we have no autopsy records to show the causes of death in the cases reported of infants dying suddenly with convulsions; but Richter, who has made autopsies in similar cases in Vienna, as mentioned above, found that the most frequent cause, in his cases at least, of sudden death was acute bronchitis complicated with, or without, acute bronchopneumonia. These infants gave few or no previous symptoms, such as fever or cough, before the fatal issue. At autopsy the bronchi were found plugged with inflammatory exudate. The death in these cases is really one of asphyxia combined with heart failure. The next most frequent cause of sudden death was acute intestinal catarrh or diarrhœal disease. In these cases death is caused by auto-intoxication with resultant eclampsia. The remaining causes of sudden death were found to be asphyxia, caused by vomiting, with consequent aspiration into the trachea of food masses, in infants the subject of intestinal catarrh or bronchitis; tuberculosis, in which a diseased gland may have broken into a bronchus; diphtheria, meningitis, intra-uterine asphyxia, in which efforts at resuscitation after birth were interrupted by sudden death; acute bronchopneumonia; and in older children sudden death may have occurred at the outset of an infectious disease. Richter found some cases of septicæmia, congenital syphilis, nephritis, anomalies of the brain or general nervous system, and congenital diseases of the heart. Aside from all the above causal agents of sudden death, there remain a few cases, now and then reported in the literature, in which sudden death occurs preceded by an eclamptic seizure or spasm of the glottis (laryngismus); and in which post-mortem examination reveals absolutely no change in the internal organs to account for the sudden death except an enlarged thymus or a lymphatic hyperplasia throughout the body, especially in the mucosa of the intestines. This cause of sudden death is more fully considered under the head of Status Lymphaticus, but it may be stated here that these cases form but a small percentage of the cases of sudden death occurring in infants or children in previous apparent health. In fact, Richter found that of the 1797 cases of sudden death of infants and children from birth to the fifteenth year, only 1 could be attributed to an enlarged thymus, with or without lymphatic hyperplasia. In this he is supported by the opinion of those who, from their experience, are competent to pass judgment upon this most important question. The physician, therefore, confronted with sudden death in a rachitic infant or a lymphatic child should be mindful not to attribute this death to external influences, but rather to diseased conditions which may be present in a particular patient.

V. THE NORMAL INFANT AND CHILD.

A good knowledge of the facts connected with the growth and development of the infant and child is essential to the understanding of diseased conditions in these subjects. Normal children vary within certain limits, as to their body-weight, temperature, pulse, respiration, and secretion of urine, in a manner similar to sick infants in the presentation of symptoms. One child may weigh more or less than another of the same age, and still be in excellent health. The physician must take into account not only the infant itself, but conditions of heredity and surroundings. There is absolutely no unvarying picture of a normal child. There are limits of variation, and these the physician should endeavor to master.

Body-weight.—During the first two or three days following the birth of the infant there is a loss of body-weight. Usually this loss amounts to from 150 to 200 grammes, or 5 to 6½ ounces (Fig. 1). It is in some infants even greater. The passage of meconium and urine, the exhalations from the skin and lungs, and the small amount of nourishment taken by the infant account for this loss. As nursing begins the weight increases until the seventh day, when the infant, under normal conditions, will have regained its original weight. On the tenth day the infant weighs 100 grammes, or 3½ ounces, more than it did at birth.

In some cases, if the infant is placed immediately after birth on a breast which secretes milk abundantly, it will not lose any, or but little, weight.

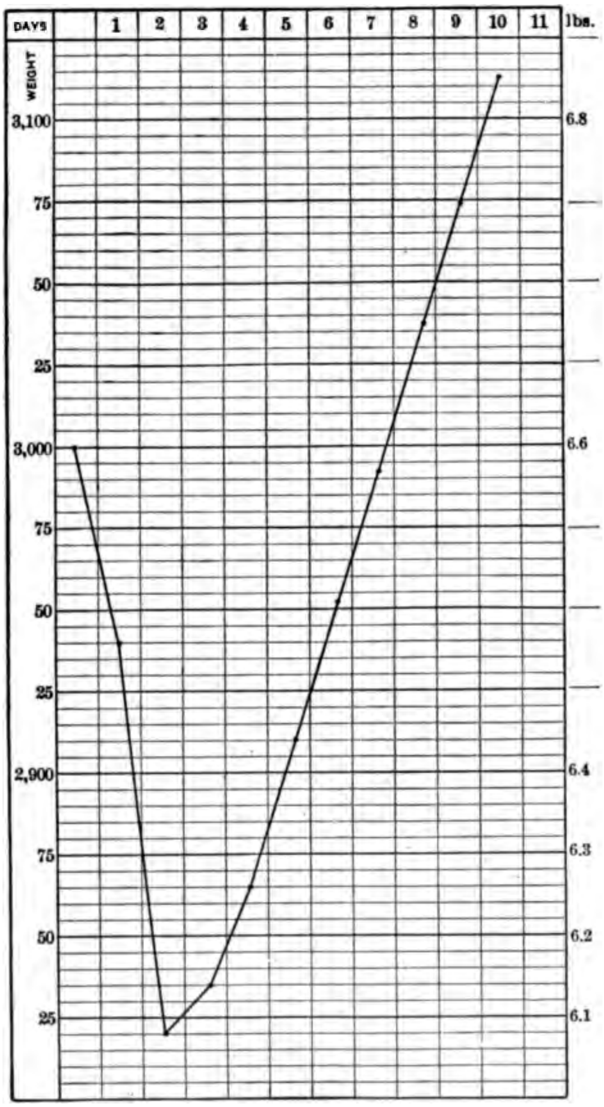
In an investigation by Gundling it was noted that many infants ceased to lose in weight after the second day, and an almost equal number on the third day. Boys lost more than girls, and the infants of multiparæ less than those of primiparæ. The average entire loss, however, was about 241 grammes. Most infants regain their original weight on the ninth day. The average infant, according to Camerer, at birth weighs 3450 grammes; and according to Budin there is a physiological loss of the amount indicated above, which is rapidly regained from the seventh day on, when the weight rises in a physiological curve throughout the remainder of infancy.

From the second week to the fourth month an infant gains 1 ounce (30 grammes) daily, or 1½ to 2 pounds a month, the latter in the first two months; from the fourth to the sixth month it will gain ½ to ⅔ of an ounce daily (17 to 20 grammes), or about a pound a month. From the sixth to the twelfth month the infant gains ½ ounce daily (15 grammes), or a pound a month.

An infant at the sixth month should have twice its initial weight; and at the end of the twelfth month a normal infant should weigh 20 to 21 pounds, or 9000 to 9800 grammes (Fig. 2).

Within physiological limits the weight given above will vary, and there are normal infants who may weigh a pound less or a pound more than the figures given. This is accounted for by vari-

FIG. 1.

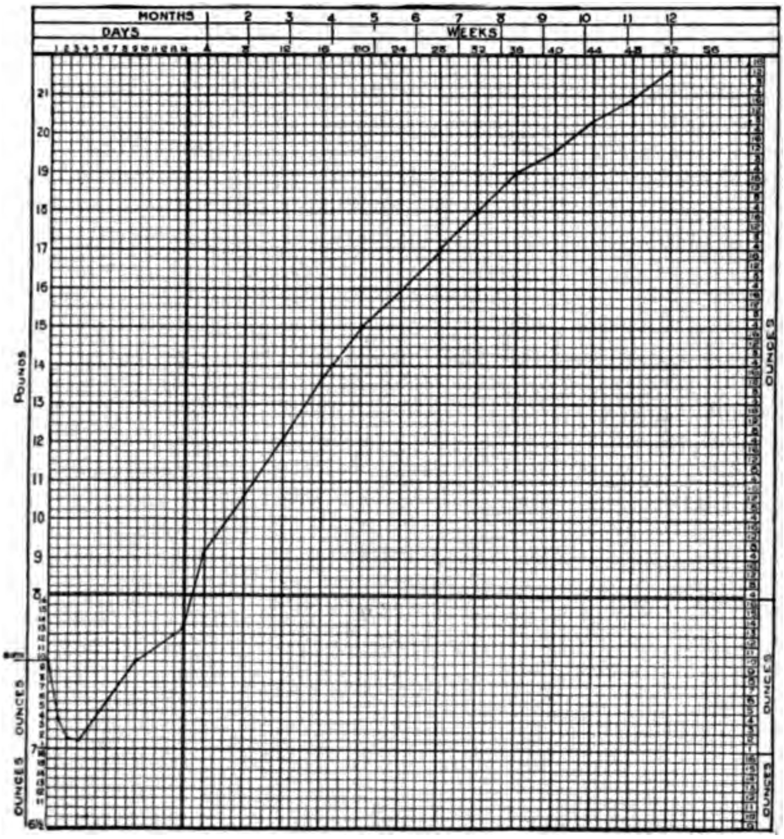


Normal curve of weight during the first ten days of life. (Budin.)

ation in the size of the skeleton, so that we cannot fix an absolute weight of 20 or 21 pounds as the normal weight of an infant at the end of twelve months, but only as an average weight.

Increase of weight differs also in artificially fed (bottle-fed), as compared to breast-fed, infants. The quantity of milk necessary to maintain nutrition is greater than in the case of the breast-fed infant. There is always the danger of overfeeding an infant on the bottle. The increase in weight is not so regular as in the breast-fed infant, as is shown in the following table:

FIG. 2.



Author's chart showing the average weight of breast-fed infants from birth to the end of the fifty-second week.

Camerer	Weeks	1—2	2—4	4—8	8—12	12—16	16—20	20—24			
	Increase	4	21	21	22	22	25	22			
	Weeks				24—28	28—32	32—36	36—40	40—52		
	Increase				13	16	16	9	12		
	Months	1	2	3	4	5	6	7	8	9	10
	Weight	3810	4430	5090	5800	6550	7180	7650	8140	8600	8880
Koplik	Months	1	2	3	4	5	6	7	8	9	
	Weight	3735		5068	5285	5518	7688	7223	8680	9021	

In the tables on pages 23 and 24 are shown in grammes not only the irregularity in the daily increase, but also the irregularity in the total weight. My own cases were examined with a view to determining what an artificially fed baby weighs if it is thriving. The figures correspond closely to those given by Camerer.

The following table shows in grammes the daily increase of weight of the breast-fed and the bottle-fed infant:

Months.	Ahlfeld (breast).	Camerer (bottle).	Koplik (bottle).
1	31	21	32.0
2	26	22	17.4
3	24	22	23.6
4	21	25	18.0
5	18	22	14.2
6	15	13	11.8
7	15	16	15.6
8	16	16	15.1
9	9	9	—

Length of Body.—At birth an infant measures from 49 to 50 cm. (19⅜–19¾ in.) in length; boys on the average having a greater length than girls. During the first year the increase in length is 20 cm. (7⅞ in.). Thus, at the end of the fifteenth year the length of the body has increased 100 cm. (39½ in.).

Head.—Herz, in a number of measurements of the head of the newborn infant, found that the

Average circumference of the head was	39.2 cm.	(15.6 in.).
Sagittal diameter	11.1 cm.	(4.4 in.).
Large transverse diameter	9.0 cm.	(3.6 in.).

The latest measurements of the head of infants, beginning at birth and extending to the twenty-fourth month, were made by Hrdlicka and Pisek, under the guidance of Dr. Chapin. These measurements are applicable to American-born infants, and are probably the most reliable we have. (See table below.)

The newborn infant has a formation of the *caput succedaneum* and in some cases of a *cephalohœmatoma*, which will be treated of in a separate section. The fontanelles, however, are of importance and may be spoken of in this connection. They are caused by the apposition of the cranial bones—the parietal, frontal, and occipital—which at first are circular, and at the points of non-contact form triangular spaces, the fontanelles. These spaces are at first closed by membrane only. At birth, or soon thereafter, the posterior fontanelle closes; the anterior fontanelle, however, remains open. The time of closure of the anterior fontanelle is of the utmost clinical importance. Kassowitz and Hochsinger contend that the anterior fontanelle grows smaller from birth to the end of the first year, and closes at from the twelfth to the fourteenth month. Elsässer and Rhodes, however, contend that, while the lateral and posterior fontanelles close during the first months of infancy, the anterior fontanelle increases in its longitudinal and transverse diameters with the growth of the cranium up to the twelfth month. Most writers, however, are inclined to accept the view of Kassowitz and Hochsinger. If the closure of the anterior fontanelle is delayed until the fifteenth month, we may look upon it as a sign of *rachitis.*

Circumference[1] *of the Head.*

Age.	Male.	Female.
1st day to end of 1st month	35.1–38.3 cm. 13.8–15.1 in.	33.4–37.1 cm. 13.1–14.6 in.
5th to 6th month	40.5–43.9 cm. 16.0–17.3 in.	41.7–44.6 cm. 16.4–17.6 in.
11th to 12th month	44.7–45.3 cm. 17.6–17.8 in.	43.7–46.3 cm. 17.2–18.2 in.
22d to 24th month	47.6–49.0 cm. 18.7–19.3 in.	45.6 cm. 18.0 in.

Respiratory Functions.

Shape of the Chest.—The ribs of some infants are quite apparent to the eye, while those of others are concealed by a panniculus of fat. The normal chest in the child has not the shape it assumes later in life—that of a truncated cone. The lateral portions are quite straight and parallel. The chest is not flattened anteroposteriorly to the same extent as in the adult. In the newborn infant the transverse diameter of the thorax is twice that of the anteroposterior diameter; whereas in the adult it is three times its length. In infants the superior border of the manubrium sterni is on a level with the midsection of the first dorsal vertebra; in the adult it is lower by a body and a half of a vertebra. The tendon of the diaphragm is horizontal in the newborn infant, and will be found to be on a level with the disk between the eighth and ninth dorsal vertebræ. A rachitic chest may be pointed at the sternum, forming what is called a chicken-breast. Some rachitic chests show a marked flaring of the lower ribs, with a lateral incurvation above this flaring; or they are flattened at the sides and deeper at the sternum. The sternum may be the top of a truncated cone, a variation from the normal state. Infants and children who have had severe attacks of bronchitis and who have some emphysema of the lungs show a marked fulness at the upper part of the chest, underneath the clavicles.

Chest Circumference.—The following measurements of chest circumference are important as showing the development of American-born infants from birth to the twenty-fourth month, and were made by Hrdlicka and Pisek, under the guidance of Dr. Chapin:

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[1] The circumference is taken just above the glabella in front and the external occipital protuberance behind.

Chest Circumference.

Age.	Male.	Female.
1st day to end of 1st month	32.0–36.7 cm. 12.6–14.4 in.	30.0–35.9 cm. 11.8–14.1 in.
5th to 6th month	39.9–43.3 cm. 15.7–17.0 in.	38.6–43.2 cm. 15.2–17.0 in.
11th to 12th month	43.4–45.1 cm. 17.1–17.8 in.	42.8–48.3 cm. 16.9–19.0 in.
13th to 14th month	42.1–47.7 cm. 16.5–18.8 in.	45.1–49.4 cm. 17.8–19.0 in.
23d to 24th month	50.7–50.8 cm. 19.9–20.0 in.	47.1 cm. 18.5 in.

The **normal number of respirations** in infants and children are as follows:

Immediately after birth	44	per minute.
From the 1st to the 2d month	24–36	"
" 2d " 5th "	20–32	"
" 6th " 10th year	20–28	"

The character of the respiratory movements in infants and children is quite shallow and irregular, especially in sleep, as compared to the adult. Respiration is of the diaphragmatic type up to the tenth year in the female child, and the eleventh year in the male, when it takes on the regular type of respiration seen in the adult.

Chemism of Respiration.—Infants nourished upon the mother's breast excrete less CO_2 than adults (Rubner, Heubner, Bendix). Thus, a baby weighing 5 kilos (11 pounds) exhales 13.5 CO_2 per square metre of surface; whereas the adult exhales per square metre 15.3 to 16.5 CO_2 (Rubner). On the other hand, the breast-fed child excretes a greater amount of water by way of the skin and lungs than the adult, on account of increased respiratory action, general activity of the infant, and its warmer apparel.

The bottle-fed baby excretes a greater amount of CO_2 and water by the skin and lungs than the breast-fed baby. This is explained by the fact that the bottle-fed infant consumes in its food a greater amount of nitrogen than the breast-fed infant.

Table of the Average Height, Weight, Head Circumference, and Chest Measurements of American Boys and Girls.

(Collated from thousands of children in various States by Bowditch, Burk, MacDonald, and Hastings.)

Years of age.	Sex.	Height.		Weight.		Head circum.		Depth of chest.		Breadth of chest.		Chest expansion.	
		In.	Cm.	Lbs.	Kilos.	In.	Cm.	In.	Cm.	In.	Cm.	In.	Cm.
5½. . .	Boys.	41.7	105.9	41.6	18.9	20.1	51.2	4.9	12.3	7.1	18.1	1.3	3.4
	Girls.	41.3	104.9	40.7	18.5	19.7	50.2	4.8	12.3	7.0	17.7	1.4	3.5
6½. . .	Boys.	43.9	111.9	45.2	20.5	20.2	51.5	5.0	12.8	7.2	18.4	1.6	4.2
	Girls.	43.3	109.0	43.4	19.5	19.8	50.3	4.9	12.3	7.0	17.7	1.5	3.8
7½. . .	Boys.	46.0	116.8	49.5	22.5	20.4	51.9	5.1	12.9	7.4	18.9	1.8	4.5
	Girls.	45.7	116.0	47.7	21.6	20.0	50.9	4.9	12.5	7.2	18.4	1.8	4.5
8½. . .	Boys.	48.8	123.9	54.5	24.4	20.5	52.2	5.1	12.8	7.6	19.4	2.3	5.9
	Girls.	47.7	121.1	52.5	23.8	20.2	51.2	4.9	12.5	7.4	18.9	2.0	5.0
9½. . .	Boys.	50.0	127.0	59.6	27.0	20.6	52.4	5.2	13.2	7.8	19.7	2.5	6.5
	Girls.	49.7	126.2	57.4	26.0	20.4	51.9	5.1	13.1	7.0	19.3	2.2	5.6
10½. . .	Boys.	51.9	131.8	65.4	29.5	20.6	52.6	5.2	13.2	8.0	20.2	2.7	7.0
	Girls.	51.7	131.3	62.9	28.5	20.5	52.0	5.1	13.0	7.8	19.8	2.4	6.0
11½. . .	Boys.	53.6	136.1	70.7	32.2	20.8	52.9	5.4	13.8	8.2	20.9	2.9	7.3
	Girls.	53.8	136.6	69.5	31.5	20.7	52.5	5.2	13.1	8.0	20.3	2.6	6.6
12½. . .	Boys.	55.4	140.7	76.9	34.9	21.0	53.3	5.6	14.1	8.5	21.5	3.0	7.8
	Girls.	56.1	142.5	78.7	35.7	20.9	53.0	5.4	13.8	8.4	21.0	2.4	6.2
13½. . .	Boys.	57.5	146.0	84.8	38.5	21.1	53.5	5.6	14.3	8.7	22.1	3.2	8.2
	Girls.	58.5	148.6	88.7	40.3	21.0	53.5	5.5	14.1	8.7	22.1	2.6	6.6
14½. . .	Boys.	60.0	152.3	95.2	43.2	21.3	54.1	5.9	15.0	8.9	22.7	3.3	8.4
	Girls.	60.4	153.4	98.3	44.6	21.3	54.1	5.7	14.5	9.0	22.9	2.7	6.8
15½. . .	Boys.	62.9	159.7	107.4	48.8	21.4	54.5	6.3	16.0	9.3	23.6	3.3	8.4
	Girls.	61.6	156.4	106.7	48.5	21.5	54.6	6.0	15.3	9.5	23.8	2.6	6.5

Table of Weight, Length, Head Circumference, and Girth of Chest from Birth to the End of the Fourth Year.

Age.	Sex.	Length.		Weight.		Head circum.		Chest girth.	
		In.	Cm.	Lbs.	Kilos.	In.	Cm.	In.	Cm.
Birth	Boys.	19.7	50.0	7.4	3.45	13.8	35.1	12.6	32.0
	Girls.	19.3	49.0	7.1	. .	13.1	33.4	11.8	30.0
6 months	Boys.	25.4	64.8	16.0	7.2	16.0	40.5	15.7	39.9
	Girls.	25.0	63.6	15.5	7.0	16.4	41.7	15.2	38.6
12 months	Boys.	29.5	73.8	21.5	9.8	17.8	45.3	17.8	45.1
	Girls.	28.7	73.2	21.0	9.5	18.2	46.3	19.0	48.3
2 years	Boys.	33.8	84.5	30.3	13.8	19.3	49.0	20.0	50.8
	Girls.	32.9	82.8	29.2	13.3	18.0	45.6	18.0	48.0
3 years	Boys.	37.0	92.6	34.9	15.9	19.3	49.0	20.1	51.1
	Girls.	36.3	90.7	33.1	15.0	19.0	48.4	19.8	50.5
4 years	Boys.	39.3	98.2	37.9	17.2	19.7	50.3	20.7	52.8
	Girls.	38.8	97.0	36.3	16.5	19.5	49.6	20.5	52.2

Circulation and Pulse.

Circulation.—According to the investigations of Vierordt, the circulation in the newborn infant is completed in 12 seconds; in the child of three years, and up to the seventh year, in 15 seconds; in the child of fourteen years, in 18 seconds; and in the adult, in 22 seconds.

Pulse.—**Its Rapidity.**—The following is the rapidity of the pulse at the various ages of infancy and childhood given by Bednar:

	Beats per minute.
Fœtus	108 to 160
First two minutes of life	72 to 94
Fourth minute of life	140 to 208
Eighth day to second month	96 to 130
Second month to twenty-first month	96 to 120
Second to fifth year	92 to 108
Fifth to eighth year	84 to 100
Eighth to twelfth year	76 to 96

The pulse-respiration ratio in infants is as 3 or 5 to 1. If we consider that the respiration in these little subjects is 30 to 32 a minute, the ratio of the respiration to pulse will be as 1 to 4 in infancy; 1 to 5 or 6 in the second year. Turning, crying, coughing, or any excitement will increase the pulse-beat 15 to 30 a minute. During sleep the pulse varies from 15 to 20 beats per minute. After the third month the pulse is more rapid in girls than in boys.

Rhythm of the Pulse.—The rhythm of the pulse has been the subject of much investigation by various observers, and the author can lay stress on the following peculiarities of the normal pulse:

(*a*) In infants and children the pulse is normally arrhythmic or irregular, both in regard to time intervals and in relation to what is known as the respiratory curve in sphygmographic tracing.

(*b*) Dicrotism is a normal characteristic of the pulse in infancy and childhood. The irregularity of the pulse in some infants and children is not very marked; in others this irregularity becomes more apparent under the influence of undue excitement. Dicrotism, although very evident and due to the great arterial elasticity in children (Landois), is never as marked as in children who are the subjects of cardiac disease, pertussis (heart strain), or acute infection (typhoid fever). On the whole, it may be said in regard to the pulse, that it is more subject to variability as a result of slight influences than that of the adult.

Body-temperature.

The temperature of the newborn infant will vary from 36.9° to 38.4° C. (98.4°–101.1° F.). The latter is exceptional. According to the studies of Lachs, the average temperature of the newborn infant varies from 37.5° to 37.9° C. (99.5°–100.2° F.). After the first bath the body-temperature falls between 1.7° and 2.5° F. Two hours after the first bath the temperature begins to rise, and reaches its original height within twenty-four hours, or sometimes later. In premature or weakly infants the temperature does not reach its original height for fully three days, and in some instances it may never reach the original height.

The body-temperature of infants shows slight fluctuations during the day which are quite normal. The maximum temperature in most cases is reached at midday or during the afternoon; the minimum, during the morning and evening. The daily fluctuations vary from 0.1° to 0.3° F. The daily fluctuations of temperature are more regular and uniform in the breast-fed infant as compared to the bottle-fed infant (Marfan). During sleep the temperature may sink from 0.3° to 0.5° F. (Alix and Vierordt). In a general way we may say that in infants and children any rectal temperature ranging from 99.3° to 100° F. is normal.

Crying, excitement, or exercise will raise the temperature in infants and children from one-half to several degrees. I have seen an instance of a boy, seven years of age, with a normal temperature, observed throughout the course of two or more years, of 100.5° F. at midday, which would rise 1° F. in the rectum after five minutes' exercise. This boy was otherwise in perfect health.

The following table of body-temperatures (rectal) is the result of the investigations of Lachs, Vierordt, and Alix:

Newborn infant	37.5° to 37.9° C.	(99.5°-100.2° F.).
5-16 months	37.4° to 37.9° C.	(99.3°-100.2° F.).
20 months-4 years	37.5° to 37.9° C.	(99.5°-100.2° F.).
5-9 years	37.6° to 37.8° C.	(99.6°-100.1° F.).

Heat Calories.—Children, according to Vierordt, produce more heat calories, per kilo of body-weight, in the twenty-four hours than do adults; thus, in children there are 130,681 calories per kilo produced as compared to the adult, where we find 39,640 calories. If, on the other hand, we accept the investigations of Rubner, in which the calories are calculated per square metre of body-surface, the infant does not use up any more calories than the adult: 1050 to 1200 as compared with 1300. The infant, for its size, therefore gives off more heat from the body-surface, and is therefore more sensitive to loss of heat than the adult.

Urine.

Physical Characteristics.—The urine up to the eighth day of life is dark in color, contains epithelial cells, leucocytes, and uric acid crystals. After the eighth day the urine is a limpid, clear, colorless fluid. The urine of artificially fed infants is somewhat darker than that of breast-fed infants, and especially is this so in any disturbance of the functions of the gut. If there is jaundice the urine may contain biliary pigment.

The urine has a resinous odor, as in the adult. The specific gravity during the first three days of life ranges from 1010 to 1012; after the tenth day, when the infant has partaken of liquid food, the specific gravity falls to 1003 or 1004. It frequently happens that the newborn infant does not pass urine on the first or even the second day of life. This is sometimes misinterpreted as due to some obstruction, either in the ureters or external genitals. From the second to the tenth day the infant voids urine two to three times in the course of the twenty-four hours. Ruge and Robin have found that at the third month the infant voids urine ten to eleven times in the twenty-four hours, passing 400 to 500 grammes in that time; at the fifth month, 400 to 500 grammes daily; from the second to the third year, 500 to 600 grammes; from the third to the fifth year, 750 grammes; and from the seventh to the tenth year, 1200 grammes daily (Parrot and Robin).

The following table gives not only the quantity of urine passed during early infancy and childhood, but shows the difference in amounts passed by the breast-fed and the artificially fed infant. It will be seen that, owing to the larger gross quantity of fluids taken into the body by the artificially fed infant, the amount of urine passed is greater than that of the breast-fed infant. The amount of urine is also dependent on the composition of the food. Camerer has shown that, as a rule, every 100 grammes of food will yield 68 grammes of urine.

Daily Quantity of Urine (Reusing).

	Breast.	Bottle.	Specific gravity.
1st day	8.4	35.8	1010
2d "	26.8	71.0	1010
3d "	40.9	135.8	1010
4th "	60.8	187.0	1010
5th "	119.1	283.0	1005
7th "	157.0	325.0	1005
8th "	208.0	406.0	1005
30th–150th day	350	Breast	1012
150th–325th "	425		
2d year	675		1012
3d–5th "	600–1200		1010–1012
6th "	1295		1012
10th "	1866		1010

The infant passes five or six times as much urine per kilo of body-weight as the adult; the child, three or four times as much.

Urea.—Urea is excreted in greater quantities by the artificially fed infant and the infant fed by a wet-nurse than by infants fed at the mother's breast. Reusing found that in the infant at the mother's breast the amount of urea increases from the first to the third day, when it is highest. The reason of the diminished excretion of urea at this period lies in the fact that there is an insufficiency of food during the first days of life. The tissues of the body are burnt up in the processes of metabolism, hence there is a diminution of weight. Inasmuch as the body is rich in fat, this is burnt first and nitrogen is saved. As a result, the nitrogen excretion in the first days is less than it is later, when sufficient food makes up for the loss of body-weight. Added to this fact of insufficiency of food, there is a paucity of fluid nourishment during the first days, causing a retention in the body of the end-products of metabolic processes. After the first few days in the newborn infant, as in all cases of starvation, there is an increase of nitrogen excreted until by means of increased food metabolism attains its equilibrium and urea is excreted in normal quantities.

Daily Amount of Urea.

	Breast-fed.	Bottle-fed.
1st day	0.06	0.33
2d "	0.26	0.40
3d "	0.52	0.67
4th "	0.50	0.55
5th "	0.78	0.65
6th "	0.79	0.61
7th "	0.81	0.88
30th-150th day	0.94	
2d year	9.87	
3d-5th "	13.9	
10th "	20.4	

Albumin.—Albumin is found in the urine, according to Flensburg, in 40 per cent. of newborn infants. He attributes its presence to the existence of uric acid infarction in the kidney at this time. Other authors contend that albumin is not present normally in the urine of infants, but if the mother has during delivery suffered from eclampsia, the urine of the newborn infant may contain albumin and casts. Czerny regards the whole question of albuminuria in the newborn as *sub judici*, inasmuch as in the cases investigated, including those of Flensburg, no mention has been made of or consideration given to disturbances of the functions of the gut or other abnormal conditions which might have been present at that time, and he is inclined to believe that if such consideration were given, it would be found that the appearance of albumin in the urine of infants is in some way connected with the disturbances of the functions of the gut.

Indican.—Indican is absent from the urine of the healthy breast-fed infant; whereas it is found in traces in the urine of artificially fed infants, even in the absence of any disease. It is especially constant in the urine of infants suffering with gastro-enteritis, and may be present in the urine of infants suffering from a number of maladies, especially forms of suppuration. It is present in the urine of infants suffering from tuberculosis, but is not pathognomonic of that affection (Zamfiresco).

Acetone.—Acetone is present in small quantities in the normal urine of infants and children, and is found also increased in quantity in the case of fevers, such as the exanthemata, or pneumonia. The amount of acetone increases in proportion to the height of the fever. It disappears or diminishes to the normal quantity with the disappearance of the fever. It is enormously increased in the urine of children during a seizure of eclampsia. It is not, however, the cause of the eclamptic seizure, as has been supposed. The cause of acetonuria is not clear. It is due neither to the hindrance of respiration nor to fermentation in the stomach or intestine; but is probably due to splitting up of the nitrogenous substances of the body, inasmuch as it is increased by a nitrogenous diet, and may be caused to disappear by an exclusively carbohydrate diet (Hammarsten).

Diacetic Acid.—Diacetic acid is not a physiological constituent of the urine, but occurs chiefly under the same abnormal conditions as acetone. There are cases in which acetone but no diacetic acid appears in the urine. Diacetic acid is often found in the urine of children suffering from some fever, such as one of the exanthemata. Inasmuch as diacetic acid is readily decomposed into acetone, it is probably an intermediate product in the oxidation of β-oxybutyric acid in the organism. Acetone, diacetic acid, and β-oxybutyric acid stand in close relationship to one another.

Urobilin.—Urobilin is absent from the urine of the breast-fed infant, but is found in traces in the urine of artificially fed infants (Giarrè and Czerny).

Dextrose.—Dextrose is found in traces in the urine of infants, as it is in that of adults. Dextrose is not found in the urine of healthy infants, and only appears in the urine of infants suffering from gastro-intestinal disturbances who at the same time may be taking food rich in glucose or maltose (Koplik).

Casts.—Hyaline and epithelial casts may be found in small numbers in the urine of the newborn infant.

Uric Acid Infarction.—Virchow has described these infarctions in the kidneys of newborn infants. They consist of red or brownish-red structures, which on section of the kidney are seen to be deposited in the pyramids of the organ, stretching from the papilla of the pyramid halfway, rarely extending to the border of the medullary portion of the organ. They exist in the kidneys, as stated, of the newborn infant, reach the height of formation on the second, and are not found after the sixth day. In the newborn infant there is a hyperleucocytosis, which is more pronounced in those cases in which the cord has been tied late. The quantity of uric acid in the urine of the newborn is much greater than it is later. In the tubules of the kidney there is an accumulation, especially in the tubuli contorti, of a hyaline substance which is the result of cell production. In this hyaline substance are deposited crystals of uric acid, and it is in this way that the infarctions are formed. The increased uric acid production is in some way connected with the hyperleucocytosis above mentioned; the leucocytes are disintegrated and uric acid thus produced. It has not been explained, however, why there is an increased elimination of uric acid with resulting infarctions at this period and not later in infancy.

VI. MENTAL AND PHYSICAL DEVELOPMENT OF THE INFANT.

It is not our purpose to enter into every detail of the development of the senses of the infant, for this would scarcely be called for in this section. On the other hand, there are certain important facts which are of great utility to the physician in his daily clinical work.

Sight.—On the second day the eyes are sensitive to light. On the twenty-first the eye will follow a light; and at the beginning of the second month the infant will notice bright colors. At the third month the infant will recognize a familiar face. At the sixth month the infant will definitely recognize its parents apart from strangers.

Hearing.—A newborn infant is deaf. This is due, it is supposed, to the blocking up of the Eustachian tubes with mucus. On the fourth day there are evidences of hearing, which develop from this time to the fifth week, when loud talking or noises in the room disturb the infant. At the sixth month the infant will recognize noises as to their varying tone.

Taste.—The sense of taste is not fully developed until the sixth month. From the fourth day, however, an infant will show a preference for sweetened, as compared to unsweetened, dilutions of milk.

Feelings of Pleasure.—An infant will show decided pleasure at the sight of playthings at the fifth month, but can hardly be said to take an intelligent interest in any object before this time.

Power to Hold the Head Upright.—The newborn infant cannot hold its head upright, and when held in arms the head will sway from side to side. The power to hold the head upright is not fully developed until the fourth or fifth month. This is important clinically in connection with certain diseases, such as amaurotic idiocy, the development of which is attended with a loss of power to hold the head upright.

Sitting.—The infant will make the first attempt to sit up at the fourteenth week; but is unable to sit upright without assistance until the forty-second week.

Standing.—The first attempt to stand without support is made by the infant at the tenth month. In the eleventh month the infant may not only stand, but even stamp its foot. Walking and standing are delayed by rachitis. In such cases the infant may even cry if placed on its feet, on account of the pain such children experience in the bones.

Crawling and Walking.—The infant will crawl on all fours in the fifth month. Attempts to walk begin at various periods, some infants being more precocious in this respect than others. The earliest attempts to walk are made in the tenth month. At the fourteenth month an infant will walk if held by the hand. It will stand alongside a chair in the fifteenth month, and in the seventeenth month a child will walk unsupported.

Laughing.—An infant two months of age may be caused to laugh in a purely reflex fashion by gentle titillation at the corners of the mouth or on the chin. An intelligent laugh, however, is not observed until the sixth month.

Kissing.—Kissing involves the act and the understanding thereof, and these are seen combined only quite early in childhood—the twenty-third month.

Memory.—True memory is observed first in the tenth month, when the infant will recognize the face of the parent after a short absence. In the twenty-first month the child will recognize its parents after a protracted absence.

Speech.—On the forty-third day the infant may articulate unintelligible sounds. At the fourteenth month it will be able to say mamma and papa; and at the end of the second year the child attempts the formation of simple sentences. In a general way, it may be said that the infant will show signs of intelligence, including sight, hearing, and vocal effort, at about the seventh month, and will first attempt to walk at the tenth month. There will be, of course, a wide variation in different infants in the development of the senses; and yet we will always recognize as pathological the vacant stare, a total lack of utterance, an indifference to bright objects, and an inability to stand on the mother's knee, or to hold the head upright at the seventh month, especially if other abnormalities, such as protruding tongue, are present.

VII. METHODS OF EXAMINATION.

Taking a History.—Beginning with a few leading questions, the physician inquires as to the sex and age of his patient, the number of children in the family, and the use of instruments, such as forceps, in the delivery of the infant. The methods of feeding the infant from the outset, are inquired into, and the success attending these methods. If the patient is in the period of dentition, the order of the eruption of the teeth is ascertained. After eliciting information in regard to any previous illness, the physician proceeds to the details of the existing affection. In the greatest majority of cases an illness in infants begins with fever, chill, cyanosis, or vomiting. One of these symptoms may be present to the exclusion of the others, or they may all be present, or the illness may be ushered in with a convulsion. The condition of the patient immediately following the initial symptom constitutes the initial stage of the illness. Fever or unconsciousness may follow a chill or convulsion, or the patient may after the initial symptom develop an eruption, cough, dyspnœa, or pain. The fever may subside in a few hours, and the temperature return to normal, with a subsequent rise, preceded by a chill, cyanosis, or a second convulsion. Older children may complain of pain, as adults do. In the case of an infant, pain in the chest or abdomen may be indicated by an increase in the number of respirations or a sighing or moaning with each effort at respiration.

The vomiting of the initial stage of the illness may not be repeated, or it may recur and form a leading feature. The nature of the vomited matter is important. It may have an acid reaction or odor, or may consist of stomach contents mingled with biliary pigment. It may be streaked with blood. In serious continued vomiting it may assume a fecal character. Vomiting may occur with the ingestion of food or independently of it.

The condition of the bowels is of importance. The movements may be numerous but of normal consistency and odor, or they may be diarrhœal and have abnormal features. The movements may be accompanied by tenesmus or prolapse of the gut. The urine of sick infants is sometimes not passed for hours. The mother will make a note of this fact. The character of the urine is next to be ascertained. Its passage may be painful. The urine may stain the diaper yellow (jaundice) or red (lithiasis); it may contain blood. Older children may be required to pass the urine. The quantity is more easily estimated in older children than in infants. With the latter we should be cautious in drawing conclusions as to the daily amount. In taking a history as above, it is essential, while eliciting the main features of an illness, not to inquire concerning unimportant details. The main features of the history should be grasped and completed in all their minutiæ.

Taking the Status Præsens.—It often happens that the infant or child is asleep during the first portion of the visit. Under that condition the respirations and pulse, with the character of each, can be noted. The posture during sleep, the expression of the face and its contour, the position and behavior of the extremities during rest, are of the greatest import. Respiration during rest is more instructive than in a condition of unrest and wakefulness. The patient should be completely undressed for examination. This is done as a routine procedure even in cases of apparently mild illness. Any eruption on the skin is thus forced upon the attention of the physician.

The Head.

The examination of the head should begin with observation of its size, whether normal or abnormally small or large. The general shape of the head and condition of the bones are of importance in reference to the presence or absence of rachitis and areas of craniotabes. The manner in which the head is held is noted, as bearing on the presence of torticollis. In Pott's disease the head is held rigidly on the spine, and in older children supported with the hands. Some infants, for instance, amaurotic idiots and those suffering from birth-paralyses or diphtheritic paralysis, are unable to hold the head upright. In forms of meningitis the head is retracted or held rigidly. The fontanelles may be normal, tense, depressed, or abnormally prominent; they may be closed prematurely or open beyond the normal period. The presence of tumors underneath the scalp should be noted. The condition of the lymph-nodes posterior and anterior to the border of the sternomastoid muscle is of clinical importance.

The Face.

The expression of the face in a condition of rest, and also when the infant or child cries, may enlighten us as to the presence or absence of paralyses. These may be localized, involving the muscles of one organ, such as the eye, or the whole side of the face may be affected. When the infant is asleep the mouth is normally closed and the infant breathes through the nose, the tongue being applied to the roof of the mouth. In abnormal states the breathing may be noisy; the cry may be peculiar, as described under Retropharyngeal Abscess; the lips may be cyanosed or the seat of rhagades or eruptions, such as herpes; the symmetry of the face may be lost, as in parotiditis or adenitis, in which there is a swelling of one or both sides of the face.

Cardiac disease in advanced stages gives a sad and anxious expression to the countenance.

Facial paralysis, either partial or complete, causes a characteristic facial expression. If the infant cries, or the child is made to smile, one side of the face remains immobile. Even in rest the angle of the mouth may be drawn toward the unaffected side of the face, as in tuberculous meningitis.

In **nuclear palsy** of the congenital variety described by Moebius and Schapringer (pleuroplegia) both sides of the face are immobile, and the face has a mask-like expression. There are no folds in the face either in the acts of laughing or crying.

Basedow's disease gives a peculiar expression to the face, caused by the prominent eyeballs, which is pathognomonic of this disease.

Hydrocephalus likewise gives a peculiar facial expression. The forehead is protuberant and overhanging. The eyeballs are forced downward, and the sclera are seen. The face proper is small as compared to that part of the head above the eyes. This is due to the large size of the cranium.

Rachitis at times causes a characteristic expression which is likely to be confounded with that due to hydrocephalus. In some rachitic infants the eyes are prominent and the sclera can be seen slightly. The orbital plates of the frontal bone being thin, the weight of the brain depresses the eyeball to a very slight degree.

Exhausting diseases, such as diarrhœa, cause prominence of the eyes, giving a very characteristic expression—the so-called hydrocephaloid of older writers.

Congenital syphilis in some cases causes a deformity of the nose, which is present at birth. The result is a peculiar angular deformity of the normal nasal curve. Looked at sideways, the bony septum is depressed; the cartilaginous septum is still intact. A very acute angle between the two results. This is similar to what is seen in destructive forms of syphilis later in life. The facial expression is characteristic of the disease.

The angle of the **palpebral fissure** is altered in conditions such as Mongolian idiocy. In this affection it is slightly oblique. In paralyses of the ocular muscles the palpebral fissure itself may be wider in one eye than in the other. The presence or absence of conjunctivitis, keratitis, nystagmus, paralyses of the orbital muscles, the condition of the pupils, are all points of importance in determining the status præsens. In diseases of the brain or its coverings an ophthalmoscopic examination of the fundus oculi should be made.

Sight.

In partial or total blindness, not only do the patients not notice objects placed in front of them, but there is in addition a vacant expression or stare. If the blindness is total, the finger will be suffered to approach the eye so as to touch the cornea.

Some infants have a tendency to hold the head to one side. This may be due to defective vision or to weakness or spasm of the muscles of the neck. In cases of defective vision the head assumes a normal position if the eyes are not focussed on any object. As soon, however, as an effort is made to accommodate, the head is inclined so as to bring the planes of vision of the eyes in accord.

Photophobia is an aversion to light, and is due to a spasm of the ocular sphincter in diseases of the conjunctiva or cornea (conjunctivitis, corneal ulcer).

Nystagmus is a series of involuntary movements of the eyeball, due to inefficiency of certain muscles, and is met with in conditions of corneal opacity, congenital cataract, albinism, infantile amblyopia, spasms, nutation or head-nodding, and in nervous states, such as amaurotic idiocy. In weakly rachitic infants nystagmus may be exhibited around a horizontal or vertical axis of the eyeball, or it may show itself in a rotary oscillation of the globe. It is made manifest in infants by causing them to focus some bright object, held slightly above and to one side of the head.

Physical Examination of the Chest.

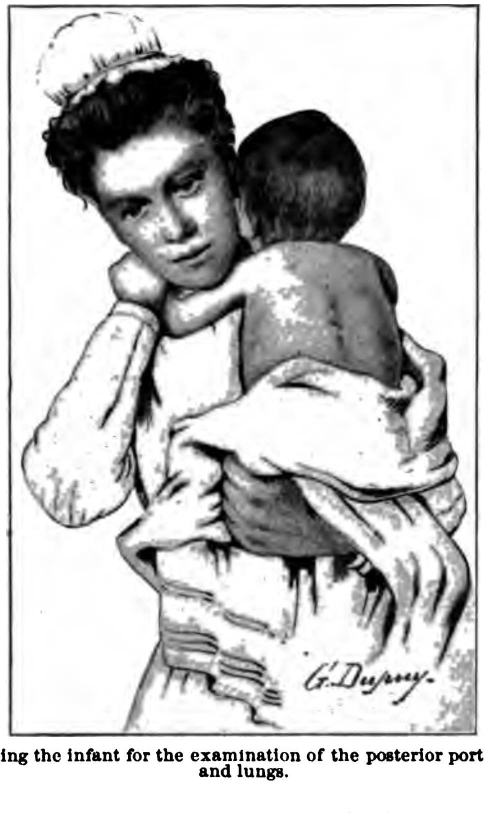
Position of the Patient.—An infant should be so held for examination that the examiner and the patient may be at ease. Being undressed, with the thorax exposed, the infant is first held by the attendant with its head looking over her shoulder, in which position the arms instinctively clasp her neck (Fig. 3). The patient so placed does not see the examiner. The spine should be straight, so that in percussing the sound is obtained on both sides under the same conditions. To examine the chest anteriorly, the infant is held looking forward, the anterior aspect of the thorax facing the examiner. If it is able to sit up, it may be examined in the sitting posture, both anteriorly and posteriorly.

With older children it is best to make an examination with the patient sitting upon a table or chair in a position convenient to the examiner. If confined to bed, the child must be examined in bed. As a rule, however, it is preferable to have the patient taken out of bed into the light.

Infants and children sometimes try to grasp the instruments of the examiner; gentle suasion will reassure them, force is never necessary.

Instruments Used.—A **stethoscope** is absolutely essential to the proper examination of the chest of an infant or child. This method is called mediate examination. We can by its means assure ourselves that the whole area of the chest has been carefully investigated. Examination by the ear—the immediate method—is uncertain. A small area of bronchopneumonia may easily escape detection in infants and children of tender age, in whom the axillæ and lateral regions of the chest should be carefully searched. Direct application of the ear to the chest is resented by infants and children, and is not a

FIG. 3.



Method of holding the infant for the examination of the posterior portion of the chest and lungs.

convenient procedure for the physician. With the stethoscope he can follow the movements of the body of a restless patient.

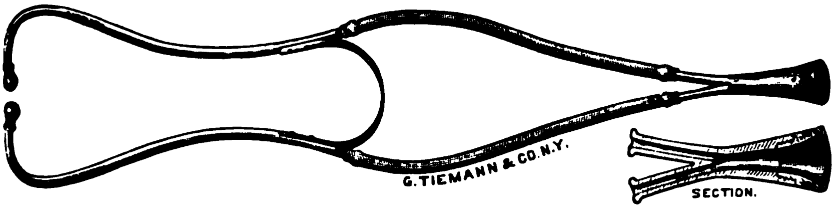
The best form of stethoscope to employ is the binaural. The instrument devised by the author (Fig. 4) has given him the most uniform results. A larger stethoscope, such as that employed for examination of the adult chest, does not differentiate the variety of sounds as well as this small instrument, and may cause pain to a restless infant, inasmuch as the chest-piece must be held too rigidly and is likely to press painfully against the chest-wall.

A **steel tape-measure**, marked off into inches and centimetres, is convenient for detecting inequalities in the size of the sides of the chest.

Methods of Procedure.—**Inspection.**—We learn by inspection the shape of the chest and the character of the respiratory movements; also, the aspect of the cardiac area, the pulsation of the apex of the heart, its force and situation.

Respiration in infants and children up to the age of ten years is of the abdominal or diaphragmatic type. The rapidity may be counted by noting the movements of the chest or by watching the rise and fall of the epigastric region in the recumbent patient.

FIG. 4.



Author's form of stethoscope. (*Archives Ped.*, Nov., 1899.)

THE CARDIAC AREA.—In some infants and children the cardiac area may be quite prominent in the absence of any cardiac disease. In rachitic infants and children this part of the chest wall may conform to the shape of the heart. There remains even in the later childhood of rachitic patients a very slight rotundity or fulness of the precordial region. If the chest-wall is quite thin, the precordial region may normally present a wave of pulsation. All these signs may be exaggerated in disease of the heart. The apex-beat is normally distinguishable. Its force and area may be increased or diminished in disease. The apex-beat may be displaced upward and outward, or inward toward the median line (conditions of effusion in pericardium or pleura).

Palpation.—Palpation, by laying the palmar surface of the hands on the chest, is hardly to be attempted with young infants and children. In these subjects the chest is so small that this method cannot mark out areas of fremitus or absence of the same. To determine its presence, it is more satisfactory to use the internal border of the hand, generally the right. The hand is held horizontally, the internal border pressing firmly against the chest-wall. Thus the slightest variations in vibration of the chest-wall can be detected. We begin above at the upper border of the chest and pass downward, comparing both sides. If the infant or child cries, so much the better. If we wish to ascertain the presence of fremitus in a baby, we may even cause it to cry. Older children may be asked to count or induced to talk. In infants and children fremitus is not so marked or useful a sign as in the adult. Normally, it diminishes in intensity toward the base of the lung. In some children it is detected in the lower part of the thorax only by careful examination. It is normally well marked along the axillary line; it is most marked along the mid-regions of the chest between the scapulæ behind. Anything which separates the lung from the chest-wall will diminish or extinguish fremitus. Solidification of lung tissue will cause better conduction and increase it.

Percussion.—It is not advantageous to use a pleximeter in examining infants and children. The index finger of the left hand is laid horizontally on the chest with firm pressure. The skin or chest-wall and finger are thus made one medium. Percussion is performed by making a hammer of the middle finger of the right hand. The force used should come from the wrist; the forearm should be immobile. The stroke is expended upon the middle phalanx of the finger on the chest-wall, and should be of a tapping character, similar to that used in striking the keys of a typewriter; there should not be a pushing motion. The force should not be great. A force equal to that necessary in the examination of the adult chest would set in vibration all the neighboring chest and abdominal organs and cavities, and would not bring out the delicate distinctions of sounds necessary to diagnosis. Moreover, to rachitic infants and young children a forcible stroke is distinctly painful.

The Abdomen.

The abdomen of an infant or child is best examined with the patient on a bed or a table covered with a soft blanket. The mother's or nurse's knees are not so satisfactory a surface for this purpose. The patient should be completely undressed.

Inspection should include the examination of the skin as to color, eruption, presence or absence of œdema, and of the abdomen as abnormally rotund or relaxed. In the latter condition we may sometimes make out the coils of gut. In diseases which exhaust the strength of the patient we distinguish between relaxed and retracted abdominal walls. A retracted abdominal wall may be tense and incurvated—the so-called boat-shaped abdomen; this is seen in meningitis. In some rare forms of septic peritonitis also the abdomen may be retracted. The pain of a colicky attack will cause the abdominal walls to be tense although not retracted. In intussusception the coils of gut or even the intestinal tumor may be seen on the surface. Ascites distends the abdomen, and when marked the rotundity is characteristic, and the skin is tense and shining.

Peritonitis causes tympanitic distention. In perforation of the gut in typhoid fever or appendicitis the tympanites is accompanied at an early stage, as in the adult, by disappearance of the liver dulness. This sign will aid us more if the liver dulness and flatness have been determined accurately in advance of any complications.

Tumors.—Abdominal tumors give an uneven contour to the abdomen. Such tumors are met in diseases of the spleen or kidney, enlargements of the liver, congenital renal cysts, ovarian tumors, or hydatid cysts.

Palpation.—We palpate for pain, general or localized, and to determine the size and position of the abdominal organs; for tumor whether of or behind the peritoneum, tumors of the liver, kidney, or spleen; enlarged glands behind the peritoneum in the neighborhood of the mesentery of the small gut; polypi in the lumen of the gut; tumors due to appendicitis or intussusception.

In palpating, we follow a certain routine, and palpate in the region of the spleen, then over the liver, and finally in the right inguinal region (appendicitis).

Ascites.—The signs are the same as in the adult.

Tympanites gives the same signs as in the adult. In newly born infants there is in rare cases a congenital weakness of the walls of the gut. Any disturbance of the intestinal tract results in immense distention, which may be distressing to the patient. Non-inflammatory is distinguished from inflammatory distention (peritonitis) by the absence of prostration or fever. There is another form of distention which precedes death in severe pneumonia or gastroenteritis. Simple tympanitic distention is seen in rachitic children, in whom the lower part of the chest is narrowed and the abdomen uniformly protuberant; in these children the distention is apparently increased by the forward curvature of the spine. Percussion gives a uniformly tympanitic note all over the abdominal area, except where fæces change the note into a dulness. There is no pain or only slight general tenderness.

Pain.—Children may locate the pain felt in pneumonia, pleurisy, or pericarditis in the abdomen. The pain may be referred to the upper part of the abdomen. The patient may complain of pain radiating to the right inguinal region, and thus in lobar pneumonia of the lower portion of the right lung mislead us into a consideration of the existence of appendicitis. In diffuse peritonitis the pain is general, but in localized disease of the vermiform appendix the limitation of pain can be made out even in young subjects. If we suspect appendicitis, it is best to examine every part of the abdomen for pain before approaching the right inguinal region.

In connection with pain and its significance, we may emphasize the fact that if the abdomen is relaxed (not retracted), showing the grooves due to the muscular parts of the abdomen—the bellies of the recti muscles, the incurvation of the abdomen just below the border of the ribs—we may assume the absence of tympanites. In such cases peritonitis is rarely present. Pain, which has no definite localization in an abdomen relaxed as above described, may be considered as of no serious import.

The condition of the abdomen in intussusception is described in the chapter treating of that subject.

Polypoid tumors in the lumen of the ascending or descending colon may sometimes be distinctly felt in the relaxed abdomen to one side of the umbilicus.

Floating kidney in children has been recently described by Comby. The methods of examination in forms of kidney tumor or displacements of this organ are described in the chapter devoted to those subjects.

Rectal Exploration.—This is always carried out in the recumbent position. By rectal examination we may establish the presence of an abscess in the right inguinal region or of great swelling of the appendix in cases in which it is bound down by adhesions below the brim of the pelvis or of ischiorectal abscess. Rectal exploration is resorted to in all cases in which we are led to suspect the presence of an intussusception. In tuberculous peritonitis also, enlarged lymph-nodes may be felt through the walls of the rectum. Kidney and ovarian tumors can in some cases be felt through the rectum.

It is not necessary to cause pain in the above procedure. On the contrary, rude examination only obscures the case. We should seek every opportunity to become familiar with the normal conditions externally and per rectum, especially in the vicinity of the right inguinal region in order to be able to diagnose abnormal states.

Examination of the Joints.

Affections of the joints are among the most frequent diseases of infancy and childhood. The method of examination of the joints should be familiar to every physician. If a mother states that her baby cries when it is bathed or diapered, we should examine the joints. In the newborn infant especially this holds true. If there is any limitation of motion, or should the extremities be limp, the joints should be inspected. In older children a sudden limp or intermittent obscure pain in a joint should receive attention at once.

To examine the joints, the patient should be completely undressed, and placed on a table. The spontaneous movements of the limbs are first observed before any manipulation of them is attempted. We may thus observe that one limb is favored by the infant, limitation of motion may exist, or there may be a marked swelling of one joint. The shoulder, elbow, knee, ankle, and other joints are systematically examined. This can be done in quite a short time if we make it a routine of every physical examination. In examining a joint we should not forget that when inflamed, it is very painful if not gently handled, and that any rude procedure, in addition to causing pain, may injure the joint.

We first inspect the joint to see whether it is swollen, or has its normal form, or shows too plainly the prominences of the bones entering into its formation. Palpation will tell whether the temperature of the surrounding tissues is raised, whether there is fluid in the joint or whether the tissues about it are infiltrated. We also examine by mild pressure with the fingers the region of the junction of the epiphysis and diaphysis for tenderness.

Mobility is tested by flexing, extending, rotating, abducting, and adducting. During such an examination we also note muscular spasm.

Joint-crepitus is a peculiar crackling, rubbing sensation found frequently in the joints of infants and children. It is detected by placing the palmar surface of the hand upon the joint and moving the extremity which enters into its formation. It has been found by the writer in children who complained of no definite joint-symptoms. It may, under these conditions, be present in many joints of the same patient. Faint crepitus is found in children who have had an attack of rheumatism.

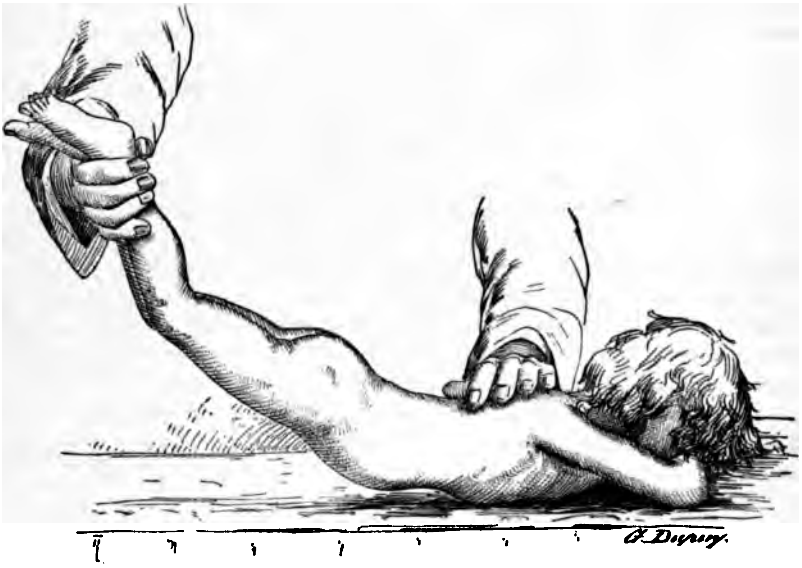
The most common affections to look for about the joints are simple luxations; syphilitic disease; osteomyelitis of a septic or infectious nature; scurvy of the joints or epiphyses in the vicinity of the joint; rheumatism, simple acute or chronic, and gonorrhœal; tuberculous joints, especially the hip; paralyses (deltoid) of muscles about a joint; deformities, as in congenital coxa vara.

The Spine.

Anatomy.—The spinal column of the newborn infant is practically devoid of natural *fixed* curves. Fehling found that there was an almost imperceptible curve backward (kyphosis) in the dorsal region and a slight lordosis in the lower lumbar region. The latter curve was more marked when the extremities of the infant were extended. The fixed curves seen in the cervical dorsal and lumbar regions later in life begin to form in the first year. They are fully fixed by the seventh year.

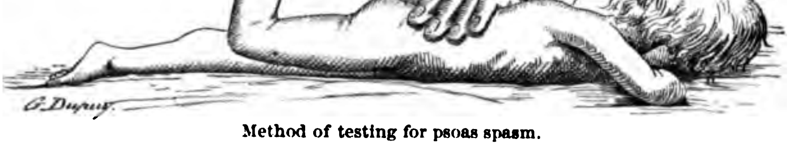
Method of Examination.—The purpose of examination is principally to discover abnormal curvatures and to test the pliability of the vertebral column. In other words, we examine for rigidity due to disease (Pott's). The patient is undressed and made to stand erect. The index finger is passed down the vertebral spinous processes, and the lines of these processes thus marked out. Any abnormal curve is thus made apparent. Painfui areas are detected by pressure or tapping along the spinous processes. If deformity is

FIG. 5.



Method of testing mobility and pliability of the spine.

FIG. 6.



Method of testing for psoas spasm.

present, it is important to decide whether this is permanent and combined with muscular spasm (Pott's) or due to rachitis. For this purpose the patient is placed on the examining table face downward. The examiner grasps both lower extremities at the ankles (Fig. 5). The palmar surface of the left hand is laid firmly on the junction of the cervical and dorsal spine. The extremities are now raised and hyperextended with the right hand. If the spine is supple and normal, it will curve backward as the pelvis is raised toward the vertical. If there is deformity due to Pott's disease, this will persist. Deformity due to rachitis will disappear under this manipulation. If the left hand is laid on the lumbar region and the above hyperextension gently carried out, first flexing the legs back at a right angle and then lifting them vertically, a distinct spasm of the muscles is felt (psoas spasm) (Fig. 6). Spinal rigidity is also made apparent by causing the child to pick up some object from the floor. Under conditions of disease the patient will hold the spine rigid in picking up the object. The hips and knees are bent, but not the spine. To test the rigidity at the outset of a meningitis, the head is raised as the patient lies recumbent. In meningitis the rigidity is such that the whole trunk can be raised by placing the palm underneath the occiput and gently raising the head.

Muscular Apparatus and Nervous System.

Form.—**Atrophy** of muscle is seen in any disease which affects the trophic centres of muscle in the cord. Such diseases are poliomyelitis, and neuritis following traumatism, diphtheria, measles, or any infectious disease. Atrophy is seen in joint-affections, especially about the hip. In the latter case, not only disuse, but a true reflex trophic disturbance is the cause of the atrophy.

Hypertrophy of muscle is seen in cases of isolated congenital hypertrophy of one limb, and also in pseudohypertrophic paralysis. In all cases of change of volume of a muscle we first inspect the affected limb and compare it with that of the opposite side if the disease is unilateral. The diseased limb is measured in its circumference and compared with the corresponding healthy limb.

Patellar Reflex.—We shall take up only that aspect of the subject which should concern the practitioner in his examination of infants and children. The minutiæ of electrical muscle and nerve reactions may be gleaned from works treating of such matters in detail.

The most common deep reflex is that of the patellar tendon. It is obtained by placing the infant in a recumbent position, supporting the thigh by placing the left hand beneath it, and raising it above the level of the body. When the muscles are relaxed, tap the patellar tendon sharply with the middle finger of the right hand. The procedure is similar to that employed in percussion of the chest. Both limbs are examined in the same manner. Children who can sit are placed on a table with their lower extremities dependent. When the attention of the patient is fixed upon some object the tendon is tapped sharply. A percussion-hammer is not necessary.

In diseases of the gray matter and of the posterior columns of the cord with trophic disturbance of the nerves (poliomyelitis, neuritis, Landry's paralysis, diphtheritic paralysis) the patellar reflex is diminished or absent.

In brain tumor and in affections of the lateral columns of the cord (multiple sclerosis, spastic disease) the reflex is increased.

The reflex is unimpaired in cerebral palsy, Friedreich's ataxia, and in cases of idiocy.

Babinski's reflex is a plantar phenomenon found in some forms of meningitis (tuberculous), and in diseases in which there is irritation or involvement of the pyramidal tracts. On irritating the plantar surface of the foot with the tip of the index finger there is a vigorous hyperextension of the great toe. Morse has shown that this reflex cannot be relied upon in children under the second year. I have had abundant opportunity to confirm this observation. As a differential diagnostic sign, the Babinski reflex is of little value, although I have observed it to be present more frequently in the tuberculous forms of meningitis than in the pyogenic varieties.

Kernig's symptom is the flexion of the leg on the thigh when the thigh is flexed at right angles to the trunk, and is found in children suffering from any form of meningitis, and in diseases such as pneumonia or typhoid fever with cerebral symptoms. The sign has the same characteristics as in the adult. In infants under one year the tendency to flex the leg on the thigh in the sitting posture is normal. In these subjects, therefore, the presence or absence of this sign possesses no significance.

Gait or Walk.—The child is undressed, so that the feet and toes are exposed, and is caused to walk to and fro in front of the physician. The gait in disease may be ataxic, spastic, paretic, or wabbling.

Ataxic gait is seen in children suffering from Friedreich's ataxia, or from tumor involving the motor centres for the lower extremities. The gait is uncertain; patients walk as if inebriated, with the feet wide apart. Incoördination of movement is characteristic of all these cases. We must in all cases distinguish between simple muscular weakness, as in pseudohypertrophic paralysis, and convalescence from acute disease, such as fevers, and a weakness combined with a palpable defect in the power of coördinate action. In cases of cerebral disease, as a rule, there is lack of coördination elsewhere, as in the muscles of the upper extremities. In these cases the coördination is tested in older children by telling the patient to close the eyes, and directing him to touch the tip of the nose with the index finger of the right hand several times in succession. In cases of ataxia there will be great uncertainty in carrying out this manœuvre. In diphtheric paralysis there may be combined with a real weakness, ataxia or incoördinate movement. If we remember that in these cases there is a neuritis, with consequent atrophy of muscle and loss of reflex, we shall not commit the error of overlooking the paralysis in our desire to account for the condition present as a simple muscular weakness the result of the illness. In these cases there may also be paralyses of the trunk muscles, causing inability to assume the upright posture. In ataxia caused by cerebral tumor there is in certain cases a crossed hemiplegia (pons tumor), with foot-clonus and paralysis of ocular muscles, which aid in the diagnosis.

Cerebellar Titubation.—In cerebellar tumor, which is the variety most common in children, there are at the outset, in most cases, disturbances of the gait or ataxia. The patients walk in an uncertain manner, generally staggering to one side. In severe forms of this disease the patients will fall to one side if not protected. The cases thus far recorded all show early involvement of the optic, auditory, and other cranial nerves, abducens paralysis, with symptoms of vertigo.

Spastic Walk.—This walk is so characteristic as not to be easily mistaken for anything else. It is found in all forms of spastic paraplegia, congenital or acquired. There is not only actual spasm, but also weakness of muscle. There are other phenomena of nervous disturbance, such as increased patellar reflex and foot-clonus. The patient seems to drag his legs in walking. Each extremity is brought rigidly forward, the toes scraping the ground. The muscles may or may not be well nourished. Electrical contractility may or may not be increased. The children may walk cross-legged (Gowers). At first there is inability to walk; later in childhood locomotion is possible. In certain forms the spasm of the extremities is so great as to keep them in constant extension at the knee; flexion in these cases can only be attained with great expenditure of force.

In infants and children who cannot walk and are the subjects of spastic paraplegia the characteristic position of the lower extremities may be made apparent by supporting the patient on the feet. In all of these cases, as soon as the toes touch the ground the reflex produces the characteristic extension of the limbs, with the toes or ball of the foot on the ground and the heel raised.

In very young infants who are the subjects of amaurotic idiocy the spastic phenomena are sometimes very marked. In these cases there are other symptoms, such as amaurosis and inability to hold the head upright, the presence of the Tay-Kingdon spot in the fundus of the eye, to aid in the diagnosis.

Limping Gait.—Joint-affections cause simply a limping gait; a study of the joint, as described elsewhere, will aid the diagnosis.

Infantile paralysis, or cerebral palsy, at the outset causes the characteristic dragging of the extremity if the paralysis is not complete. Infants in whom there is a complete loss of power in one or both lower extremities give a history as follows: The infant may have been able to walk or stand; the attack suddenly deprives it of the power of motion. There is a limp extremity on one or the other side, with rapid atrophy of muscle and loss of reflex. In cerebral palsy there is no atrophy and the tendon reflex is present.

The methods of examining the mouth and special organs will be taken up in the chapters devoted to their diseases.

VIII. MANAGEMENT AND HYGIENE OF THE NORMAL INFANT.

Taking the Infant from the Mother at Birth.—As soon as the infant is born and pulsation in the cord has ceased, the cord is tied and the physician places the newcomer in the care of the nurse. The tying of the umbilical cord should be performed rapidly, and the nurse, for this purpose, should have at hand a piece of sterilized tape or broad binding-silk and scissors which have been boiled in water and then carefully wrapped in a clean towel. It is not necessary to use silk which has been soaked in antiseptic solutions, such as carbolic acid, for the infant is peculiarly susceptible to these drugs. I have recently seen an infant whose cord was tied with silk saturated with a very strong solution of carbolic acid who, within a few hours after birth, showed signs of the action of the drug on his economy. The sterilized tape and scissors should be in readiness for the physician, as searching for the tape or scissors causes an inexcusable delay. A warmed piece of soft blanket is wrapped about the infant at once. As is well known, the infant at birth cries lustily; nature intends that it should be so at this time in order that the lungs may be filled with air.

Tying of the Cord.—The physician should tie the cord, as has been stated, with a piece of sterilized tape or broad binding-silk, about an inch or an inch and a half from the body, after the pulsation of the cord has ceased, unless some feature in connection with labor indicates a more rapid procedure. After the initial bath the cord is inspected to see that the first ligature is still intact. Ahlfeld reties the cord close to the abdominal wall, though this seems to be unnecessary. If the ligature is still in place and there is no hemorrhage, the stump of the cord and the surrounding tissues are washed with strong alcohol, and a sterilized dry gauze pad with inclosed absorbent cotton is folded over the umbilical stump. This is held in place with a clean body-binder. The first dressing is not removed until the stump of the cord has fallen off and the umbilicus has healed, unless there is some indication for its renewal, such as the soiling of the dressing by the urine of the infant (Ahlfeld).

Another method of dressing the cord is to form a pad of absorbent gauze four or five layers thick, about three inches square, cutting a small opening in the centre. The stump of the cord is passed through this opening and the gauze folded over the stump. The dressing is secured with an ordinary body-binder. This dressing, also, is not disturbed unless it is soiled by the urine of the infant.

Stump of the Cord.—The stump of the umbilical cord dries up and falls off from the sixth to the tenth day. It may fall off as early as the third or as late as the fourteenth day. In premature or weakly infants this process is delayed. Even in healthy infants a delay may occur which has no pathological significance. When the cord drops off there is a flat, granulating surface left, which cicatrizes, and after a time takes on the appearance of the neighboring skin. Occasionally, however, the site of the stump takes the form of a small pea-like body; this is pathological and will be discussed elsewhere. Normally there should be no protrusion of the umbilicus, even when the baby cries. The drying or mummification of the stump of the umbilical cord is a purely physical process, and depends more or less on the dryness of the dressing on the stump of the cord. When the stump of the cord remains dry, but few bacteria are found in the tissues; if, however, as in very exceptional cases, moist gangrene of the stump takes place, staphylococci and streptococci in large numbers appear in the stump and the immediate vicinity. The stump of the cord is thrown off by a sort of reactionary inflammation at the point of juncture of the amnion sheath of the cord and the skin. A few hours after birth the capillary network in this vicinity is seen to become congested. The amnion first separates, then the arteries, and finally the vein, leaving a granulating base at the umbilicus.

First Bath.—The question has been much debated as to whether an infant should be bathed immediately after birth or whether the body should be simply anointed with vaseline or olive oil, wiped off, and not bathed until the stump of the cord has fallen off. Whatever objection there is to bathing premature infants, this cannot hold with infants at full term. The bath is cleansing. The lochial discharge of the mother if allowed to remain in contact with the skin is apt to decompose, and a source of infection is at once presented.

The most convenient form of bath-tub for the infant, if it can be obtained, is that constructed of rubber sheeting. It obviates placing under the infant any blankets, as must be done in a bath-tub made of metal. These bath-tubs are constructed so as to have a certain height from the floor convenient for the nurse. They hold heat better than the metal bath-tub.

The temperature of the room in which the newborn infant is bathed should be 70° to 72° F. The bath-tub should be situated, if possible, near an open fire, to insure warmth.

At birth the infant is covered with a white substance, the vernix caseosa, which must be carefully removed, and to this end the body is anointed with vaseline or olive oil, the latter being preferable to vaseline, which may irritate the skin. When the infant is anointed it should be exposed part by part only, in order to guard the body-temperature from being rapidly reduced, and care should be taken not to displace the ligature or roughly handle the stump of the cord, lest hemorrhage result. The first bath is, therefore, a scientific function; it cleanses and protects the infant from present and future auto-infection. The water in which the infant is bathed should be boiled, in order to destroy any extraneous source of infection, for, as will be seen later, the bath-water has been the cause of epidemics among the newborn, especially in hospital service. In private practice this danger does not obtain to the same extent as in institutions.

The function of bathing the infant should be performed rapidly, and at the same time in a painstaking and gentle manner. The water of the bath should be 100° F., and some additional warm water should be at hand in order that the temperature of the bath water may be maintained at this point. The infant is placed in the bath, and with glycerin soap rapidly washed, and lifted out and placed in a warm blanket. The depth of the water in the tub should be just enough to cover the body. The head is supported above the water by the disengaged hand of the nurse. The infant cannot then slip out of the arms of the nurse. While in the bath the infant is constantly but gently rubbed, and when taken from the bath should not be blue or in the least chilled. Drying the infant is best performed on the knees of the nurse, part by part, so as not to expose the infant's whole body at one time. The cord is dressed as above described and the binder applied. All clothing, including the binder of the newborn infant, should be made of soft flannel or pure wool.

Daily Bath.—There has been some discussion as to whether an infant should be bathed daily, after the first bath, before the separation or falling off of the stump of the umbilical cord. It has been demonstrated that infants who are not bathed in the first week lose less in weight than those who are bathed. It is best, therefore, in order to avoid infection of the umbilical wound, to favor mummification of the cord, as well as to conserve the weight of the infant, not to give a full bath, after the first bath detailed above, until the umbilical wound has healed and the stump of the cord has separated. When this has taken place the infant is bathed daily; up to that time it is washed twice daily, with a view to cleanliness. If the dressing on the umbilical stump has become soiled with urine, or otherwise, it is changed; but unless this indication exists the first dressing is left undisturbed.

The best time for the bath is in the forenoon, one hour after nursing. The temperature of the water of the infant's bath should not be below 99° or 100° F. during the first ten days; 95° F. during the first month of infancy; and 90° F. after the sixth month. It has been proposed—on grounds which are somewhat obscure and not founded on physiological facts—to harden the infant by means of a gradual reduction of the temperature of the bath-water until, even with an infant below one year, the bath-water is quite cool. Such a procedure does not harden the infant; on the contrary, it has been shown that it is directly detrimental to his growth and well-being. Delicate infants, even those born at full term, may by such a process of hardening contract a bronchitis, or even some more dangerous affection of the lung.

The details of the daily bath are much the same as those described with the newborn infant. The use of a sponge in bathing is not cleanly or desirable. A soft piece of linen or muslin or so-called washcloth is much to be preferred, as it can be easily cleansed and boiled. After the bath the infant is taken from the water and placed in a soft, warm blanket or bath robe, carefully dried and powdered. Powder is applied to the axillæ, groins, buttocks—where surfaces come in contact. The general surface of the body is not powdered unless some indication exists.

Premature Infants, and Infants who are Under weight.—Infants born prematurely or those who weigh six pounds or less, even though born at full time, should not be bathed as above described, but are best washed part by part with warm water once a day until the weight has reached the normal limits. These puny infants are particularly susceptible to reduction of temperature. In fact, the rectal temperature in such infants is always low, and any bath, even a warm one, will reduce the temperature still more and may result in serious chilling of the body.

Hardening.—It will be seen from what I have said that I do not believe in the so-called hardening process as applied to children. I have seen children, whose mothers took a pride in bathing them with cold water, who remained pale, stunted in growth, nervous, even with a flabby musculature, notwithstanding a daily regimen of cold water which was intended to have a tonic effect, both on the general nervous system and physical development of the child. In my experience I have rarely found, at least in this climate, that any other temperature for bathing was indicated but that which has been mentioned above. A very excellent guide as to the proper effect of any form of bathing on an infant is the so-called reaction in and immediately after the infant is taken out of the bath. In the bath and after bathing the infant should be warm on the surface and present a ruddy appearance. If during or after a bath the infant is cyanosed and the surface of the body is cool, we will conclude that the bath, at whatever temperature it is given, is not adapted to the infant.

Eyes.—In a maternity service, where numbers of women are delivered and there is danger of one infant being infected by another, it is customary to instil into each eye at birth a drop of a 2 per cent. solution of nitrate of silver. This is done as a prophylactic measure against gonorrhœal ophthalmia, a disease which has been proved to be a great etiological factor in the causation of blindness. In private practice, however, this is scarcely necessary (see Ophthalmia Neonatorum), especially if we are acquainted with the condition of the mother and no vaginal discharge has been present previous to labor. If, however, there has been a vaginal discharge before labor, it is well either to apply the Créde method and instil a drop of a 2 per cent. solution of nitrate of silver into the eye, or to carry out the prophylactic measure of Kaltenbach, described in the section on Blenorrhœal Ophthalmia.

The eyes during infancy need no attention other than that customary in the adult—cleanliness. Any slight discharge from the eye indicates a conjunctivitis. The nearer this conjunctivitis occurs to birth, the more we should be on guard for detection of a gonorrhœal process. It is always wise, therefore, as soon as any secretion of pus is detected in the eyes of the newborn infant, to examine this pus for microörganisms of a specific nature. Any swelling of the conjunctiva or the lids should put us on our guard against gonorrhœal infection.

Method of Taking the Body-temperature of the Infant.—The temperature of infants and children is always taken in the rectum ; but if the child is above five years of age we may, under certain conditions, take an axillary temperature. Some children are terrified at the sight of a thermometer ; others have an innate modesty, which it is the duty of the physician to respect, and which precludes the taking of a rectal temperature. If the indication is not pressing, therefore, an axillary temperature may be taken in older children in the same manner as in the adult.

It is well in dealing with children to teach the parents how to use the thermometer. In this way each child may have its own thermometer, whether it is used in the rectum, the axilla, or the mouth. This is not only convenient for the physician, but is entirely proper, especially as applied to children, for thermometers cannot be thoroughly disinfected, and it is certainly objectionable for a physician to go from one little patient to another, introducing the same thermometer into the rectum.

In introducing the thermometer into the rectum, the infant or child should be laid on the side. The bulb of the thermometer is anointed with vaseline or olive oil, the buttocks are gently separated with the fingers of the left hand, and with the right hand the bulb of the thermometer is carefully insinuated into the rectum. The infant or child is continued on the side for at least three minutes. The thermometer is then removed, and after reading the register the physician should carefully cleanse the thermometer, before proceeding further, with a piece of cotton first, then with a fresh piece of cotton moistened with ether and then alcohol, and finally with a 1 : 2000 solution of corrosive sublimate or a 0.5 per cent. solution of formalin. In private practice this paraphernalia is not always at hand, and the physician can see at once the utility of teaching the parents to have a thermometer in the house for the use of the child rather than that he should imperfectly cleanse his own thermometer and use it on another patient. In children's hospitals this question of individual thermometers is of great importance, and no children's service can be conducted without danger of infections arising unless each patient has his or her own thermometer.

Temperatures should be taken in mild cases of illness and in convalescence three times daily; in protracted and serious illness, such as pneumonia or typhoid fever, every three hours throughout the twenty-four.

Diapers.—The diaper should be made of an absorbent material, such as well-washed soft muslin or linen, and should be about two yards square. It is first folded in the middle, then in three-cornered fashion, refolded, and thus applied to the infant. A diaper should not be covered with a rubber protection except during travel, inasmuch as under these conditions the diaper becomes, if moistened, a species of poultice and intertrigo results, as well as eczematous eruptions of the buttocks. Diapers should be applied warm and dry. A moist diaper will sooner or later cause a skin eruption. A diaper moistened with urine should not be dried and used again on the infant, for by this method the salts of the urine are crystallized in the meshes of the diaper fabric and will irritate the skin. Diapers when soiled should be placed in a covered utensil sold in the shops for this purpose. Before washing the diaper the excess of movement should be removed. Diapers should be boiled in an alkaline solution, generally of soda, and should be washed by hand and not with the mandril, otherwise the fæces and discharges cannot be removed thoroughly.

After a movement the child is dried gently with a piece of soft linen, sponges not being used, carefully powdered, and a new diaper applied. Diapers, if soiled, should not be put into a disinfecting solution. On the contrary, there is a positive objection to this, as diapers permeated with drugs may cause irritation of the skin of the buttocks. After changing the diapers, the nurse's hands and finger-nails should be cleansed with brush and file. This toilet of the hands and finger-nails is very important, even with breast-fed infants, since the neglect of this function will result in a contamination of the breast nipple or food with fæcal bacteria. Even the infant's own fæces may cause serious intestinal disturbance if reintroduced in the above manner into the stomach and intestine.

Care of the Genitalia.—The care of the genitalia in male and female infants is quite important, and it is surprising to see how such a simple matter is neglected by the mother and nurse. In female infants and children during the bath the labia should be washed, gently separated, and the parts beneath laved with water. After the bath these parts should be carefully dried, but not powdered. It is a very common practice to powder the parts beneath the labia majora in female infants. This custom causes considerable irritation around the introitus vaginæ, as a result of the powders settling on the parts. If these parts are not powdered, but simply dried after the bath, they will remain in a normal condition, and an accumulation of smegma will be avoided.

In male infants the prepuce should be retracted daily and the parts bathed with ordinary water. In this way accumulation of smegma, and balanitis will be prevented. It is not necessary to use medicated solutions, such as boric acid, for this purpose. In boys the scrotum, buttocks, and adjacent parts should be powdered.

Play; Fondling.—It must not be forgotten that the average infant's stomach is easily upset, and that any kind of pressure on the abdomen is often a very effective way of emptying the stomach. After feeding, therefore, the infant should lie quietly in its crib and not be handled or fondled. Unless this rule is followed, vomiting after nursing will quite frequently occur.

It should be remembered that too much play is apt to tire an infant as much as it would an adult. Infants who are played with and fondled to excess are tired, restless, irritable, and sometimes do not sleep. There is no rule to be applied, but moderation is to be followed in these things as in all others concerning the infant's pleasure.

Sleep.—An infant in perfect health spends most of the time in sleep when it is not nursing. Unless its attention is engaged by others, it will not play in the early months of infancy. After nursing, an infant falls asleep, generally on the breast. Therefore, if an infant cries or is restless after nursing, there is something at fault. Older children should sleep in the afternoon for one hour, after the midday meal. This should be especially insisted upon with children who have a nervous temperament. If such children do not attain an early habit of sleep in the afternoon they will be restless at night, and finally develop symptoms of neurasthenia. Children should not be allowed too much intercourse with adults, as this is also apt to have a deleterious effect. Children should play with children. Adults should limit their play and contact with children as much as possible.

Bed.—The best bed for the newborn infant is one in the form of a bassinet. The infant certainly should not sleep in the bed with the mother or nurse, for, aside from the danger of so-called overlying, the infant is liable to become infected with the discharges of the mother; and in a breast-fed infant there is always a temptation to give the breast to the child at night whenever it is restless. Bad habits therefore result. Aside from this, an infant will be restless unless trained to sleep in its own bed.

The mattress of the bed should consist of a hair cushion protected by a rubber draw-sheet. Over this is placed a bed-pad, and over this the bed-sheet. After the fourth month an infant may be placed in a crib. For restless children cribs are made with high sides, so that they may not fall out. Rocking bassinets or cribs are undesirable. An infant accustomed to such a rocking-crib or cradle will not fall asleep unless rocked, and the mother or nurse becomes a slave to the crib. If a baby in early infancy cries without any apparent cause just as it is placed in the crib from the mother's or nurse's arms, it is best not to take it up immediately, for, unless this habit is broken in early infancy, an infant will refuse to be pacified unless taken up several times in the twenty-four hours.

The physician may be consulted concerning the pillow for the infant, as to whether it should be made of hair or down-feathers. It is well for the young practitioner to know that a pillow made of the finest curled hair is really more comfortable than a down-pillow. When placed under the infant's head, the pillow should reach well beneath the shoulders, so that the head and shoulders are supported together. The custom of not using the pillow for the infant allows the head to come in direct contact with the mattress, a very uncomfortable position, and one which inevitably results with careless mothers or nurses in a slight erosion at the back of the head, over the occiput.

So-called pacifiers made of rubber or muslin should never be used in the nursery. They are undesirable and unnecessary, and if not used will not be in demand.

Nursery.—The temperature of the room in which the infant passes its days should be carefully maintained at from 68° to 70° F. Variations in the temperature of the room not only chill the infant, but interfere with its growth and nutrition. Drafts are reprehensible. The air of the room should have no odor, and we should ventilate indirectly from another room which is warmed. Incense should never be used to cover up an odor. The nursery should be well lighted, as well as capable of ventilation. An open fireplace aids the ventilation considerably, and in damp weather dries and warms the atmosphere as well as ventilates the room.

The floor of the nursery should be made of hard wood or painted and covered with rugs. Carpets are not hygienic. They must be swept *in situ;* whereas rugs can be taken out, dusted, and aired. The crib should be protected from the open window by means of a screen. During infancy, up to the twelfth month, the temperature of the nursery, both day and night, should be kept at the same point. There is no reason why the temperature should be lower at night than during the day, as is customary in the sleeping-room of the adult. When the infant is in the open air, the nursery should be thoroughly ventilated for at least an hour a day. With premature children, however, we must be more careful and keep the temperature at a slightly higher point than the above. Or, if we have the room at 70° F., such children should be aided in maintaining the body-warmth by means of warm bottles placed underneath the blankets in the crib, but not necessarily close to the body.

Open Air.—The infant may be taken into the open air three weeks after birth in the summer season and four weeks after during the winter, early spring, and fall. I have consistently advised that four weeks after birth, if the weather is not too cold, the newborn infant may be allowed an outdoor airing. I have seen no bad results follow from this advice. If the weather is exceedingly cold, common sense would dictate that all infants should be kept indoors. After this time a daily open-air exposure is always allowable in good weather, provided the infant be warmly clad, especially in the winter time, so as to run no danger of chilling. If an infant shows a tendency to be easily chilled when taken into the open—which can be judged by observing the color of the face and hands—warm bottles should be placed underneath the covers of the baby carriage.

Infants should be protected from the direct rays of the sun, inasmuch as they burn and tan very readily. Tanning of the skin, or sunburn, is not necessary to the health of the infant. A physician will frequently be asked whether sleeping in the open air is injurious to the infant. It certainly is not, provided the infant is well protected in the manner described above. Some infants fall asleep immediately on coming into the open. We could scarcely keep such infants awake, and nature simply indicates to us in this way that the open air is a tonic to the general nervous system. In large cities, both in summer and winter, the face should be protected by a veil when the infants are taken into the open. In the country this is especially necessary if mosquitoes and flies are in the vicinity. Children who are running about should not wear short stockings if the locality is infested with mosquitoes or insects. There is nothing particularly hygienic in the custom of wearing short stockings, and it exposes the children to the danger of infection, not only by mosquitoes (malaria), but by dangerous insects, such as spiders.

Clothing.—**Body-binder.**—It is customary to provide the new-born infant with a body-binder made of soft, white, thin Shaker flannel, five inches wide and sufficiently long to pass two or three times around the body. It should be secured with strings, and not with pins, nor should it be sewed on the body. It is useful at first in retaining the dressing of the cord in place, and later on in supporting the umbilicus during straining or crying. The binder is discarded when the infant first makes attempts to stand. This usually occurs at the seventh month. The binder then loses its utility, inasmuch as the umbilical opening is naturally closed and supported by the muscular action of the recti muscles. It is customary, however, to substitute for the binder, when it is discarded, a so-called knitted flannel band, sold in the shops for this purpose.

Clothing.—The clothing of the infant should consist of a chemise of wool next the skin. Over this there should be a loose garment, either wool or flannel, reaching from the shoulder to below the feet, and sufficiently long to allow it to be folded upward. Garments should not restrict the chest in the old-fashioned way. The chemise should be made of gauze weight in summer and slightly heavier in the winter. Some infants cannot tolerate the contact of wool with the skin, because it causes an eruption of sudamina; in such cases it is well to place between the skin and the woolen garment a fine-linen chemise.

Skin.—The precautions which should be observed in drying the skin after the bath have already been mentioned. Dusting-powders that contain perfume should be avoided. Dusting-powder is applied with a puff of absorbent cotton in preference to a powder-puff. This absorbent cotton can be thrown away and a new pledget used at each dressing. To prevent caking, any excess of powder should be removed.

If the skin is subject to sudamina in the summer, a handful of bran is added to the water, or, what is preferable, the bran is put into a gauze bag, moistened and expressed in the water of the bath until the water becomes turbid. Salt water irritates the skin of these infants and should not be used.

Mouth.—It was formerly customary to wash the mouth of the infant thoroughly either after each feeding in bottle-fed infants, or two or three times daily in breast-fed infants. There is really no scientific indication for doing this if the nipples of the bottles used for artificially fed infants are kept scrupulously clean; and the mother's or nurse's nipple for the breast-fed infant be cleansed with a solution of boric acid before and after each nursing. The mouth of the breast-fed infant especially should not be frequently washed. If the breast nipple is kept scrupulously clean, sprue or stomatitis will be avoided. Before the eruption of the teeth, the natural secretions of the mouth are quite sufficient to keep the mouth clean. After the teeth have appeared they may be kept clean by washing once a day with a piece of lint or cotton moistened with boric acid solution. In the morning, after the bath, the mouth of the infant is carefully washed with a piece of absorbent cotton wrapped around the finger and moistened with boric acid solution. No force should be used, and no hard pressure exerted against the roof of the mouth especially, as in this way ulceration may result.

Before introducing the finger into the mouth of the infant, the nurse should thoroughly cleanse the hand. In order to avoid the introduction of sprue into the mouth, the bottle nipples should be boiled once a day for ten minutes in a soda solution, and cleansed carefully with hot water after each nursing. In the intervals of nursing the rubber nipples are best kept either in a glass-covered jar or in a piece of absorbent gauze. It is well not to keep them in a solution of boric acid, as this is apt to become contaminated.

It has been maintained by some that washing the mouth of the infant nursing at the breast is prophylactic against infection of the breast by bacteria of the infant's mouth. Aside from the fact that the bacteria which exist in the mouth of the newborn and young infant, before the eruption of teeth, are not pathogenic, no one has proved that they are capable of causing breast abscess. Epstein has shown conclusively that washing the mouth of infants is productive of infectious ulcerations of the mucous membrane of the buccal cavity, as well as the means by which extraneous infections, such as gonorrhœa and sprue are engrafted on the mucous membrane.

In the newborn the production of buccal ulcerations as a result of a too diligent toilet of the mouth is not without great danger. It has been long acknowledged that bacteria may gain access to the circulation through these ulcerations and thus cause general sepsis.

IX. THE ADMINISTRATION OF DRUGS AND OTHER METHODS OF THERAPY.

Children should receive drugs in an agreeable form, although some may take nauseous drugs with apparent indifference. Bulky mixtures or drugs which are apt to upset the stomach should not be prescribed. The author recently saw a severe enterocolitis set up by a cough mixture containing antimony. Drugs should not be administered in pill form to infants or children. Tablets are a ready means of administering certain drugs. They can be crushed and given in a teaspoonful of some indifferent fluid. Powders are also easily taken. They are put in a spoon, some fluid added to form a mixture, which is then administered. Quinine is given either in syrup of yerba santa or in chocolate powder and water; or the child is given a piece of chocolate to eat, and then is caused to take the quinine immediately afterward. A child should never be forced to take a medicine. Much harm is done in this way. Certain drugs, such as opium in the form of the simple tincture or morphine, are not given to children under the age of two years. Atropine, of late advocated in cholera infantum, should not be given to infants and young children. They bear this drug badly. Jaborandi is badly borne, as is also apomorphine. Camphor is a very good cardiac stimulant. It is useful in collapse, but must be given cautiously in cases in which there is diarrhœa. In the latter disease the camphor is apt to irritate the stomach and gut. The coal-tar series, such as antipyrin, antifebrin, and phenacetin, are powerful depressants. In those cases of fever in which it is not possible to give baths to lower the temperature we are sometimes forced to administer these drugs. It is then well to combine with them some caffeine.

If a child or an infant refuses to take a drug, it may be put in a teaspoon, the spoon held horizontally to the lips, and when the mouth is opened the spoon carried far back into the mouth and tilted. The spoon is held in the mouth until the act of swallowing, which must inevitably take place, is completed; the spoon is then withdrawn. If this manœuvre is thus carried out, the fluid will not be rejected. Holding the nostril closed, and thus forcing the child to open the mouth, is bad practice. Patience and suasion can accomplish as much in most cases.

Digitalis is not given continuously, but is administered for two or three days, and when the pulse begins to show signs of lessened frequency its administration is suspended. Alcohol is well borne by children. I do not hesitate to administer it in cases of nephritis if the heart is weak. In the gastro-enteritis of nurslings the stomach is very intolerant of alcohol. It should not be given except in very severe cases with great prostration, as the vomiting is apt to be aggravated. Much has been written concerning antipyresis and antipyretics in the treatment of the diseases of infancy and childhood. The young practitioner can feel assured that high temperatures are well borne by infants and children. A temperature of 106.5° F. (41.3° C.) in an adult, although of short duration, would cause great alarm, and rightly so. On the other hand, such a temperature in an infant or child does not necessarily threaten life, nor is it incompatible with recovery. A convulsion in some children is the direct result of a rise of temperature. Such a convulsion will not necessarily lead to others nor to epilepsy. The heart and kidneys bear long-continued high temperature well in comparison with those of the adult. The most trivial causes will cause a rise of a degree or two in the temperature of an infant or a child. Taking all these idiosyncrasies into consideration, it may easily be understood by the student and young practitioner why it is essential that methods of therapy should be modified before they can be applied to infants and children. A reduction of temperature from 104° to 102° F., even if it can be accomplished by a coal-tar derivative, does not cure the patient. Some diseases, such as measles, scarlet fever, pneumonia, and a host of others, run a course of high and low temperatures extending over a certain space of time. If an infant or child is attacked with convulsions following every acute rise of temperature, the parents should be warned of this fact. In these cases, as soon as a rise of temperature is noted, it should be combated by every means in our power. Reduction of temperature in such children at the outset of a disease is of the highest utility. It saves the nervous system from the shock of a convulsion. Hydrotherapy is, as a rule, the safest and most satisfactory antipyretic measure at our disposal.

The dosage of drugs for infants and children has received much attention. In practice we judge more by the action of a remedy than the quantity administered. The initial dose should be small. Infants under a year receive $\frac{1}{20}$th of the adult dose, and at the age of one year $\frac{1}{10}$th of the adult dose is safe. At the fifth year $\frac{1}{5}$th, and at the tenth year $\frac{1}{2}$ the adult dose is the rule. These figures are not absolute. Nitroglycerin if given in doses of less than $\frac{1}{250}$th of a grain has scarcely any effect on children five years of age. On the other hand, strychnine may be safely given in quantities of $\frac{1}{250}$th of a grain to infants, and $\frac{1}{150}$th of a grain to children two to three years of age. It will be seen that if the hard-and-fast rule of division of doses according to age were followed, these drugs would necessarily be given in much smaller dose, and their action would be correspondingly inefficient.

Hypodermic administration of drugs to infants and children presents nothing peculiar, as compared with the same method applied to adults.

Hydrotherapy.

The practice of hydrotherapy as applied to the adult must be somewhat modified before it can be carried out with the infant or the child. The reason for this is that the infant or child does not react so readily and cannot bear sudden changes of temperature so well as the adult.

The Sponge Bath.—A rubber sheet is placed on the crib, and over this one layer of a small blanket; the patient is then placed nude on this blanket and covered with another blanket. There is thus no undue exposure. A small basin of water at 80° to 85° F., with a dash of alcohol, is now brought alongside of the crib. With a small sponge or piece of soft folded linen the parts of the patient are sponged; first one arm, then the other, then the trunk, and finally the lower extremities. As each part is exposed, the rest of the body is kept covered. This procedure is repeated until the body has been sponged for five or ten minutes. This method of hydrotherapy is especially suitable in acute rises of temperature of short duration and in mild cases of continued fever in which the temperature does not rise high.

Cold Chest Compress.—Three layers of linen are cut so that they will envelop the trunk from the clavicles to the umbilicus. The general shape should be that of a shirt deprived of arms and open at the sides. On the outside of this linen compress there should be a compress of Shaker flannel cut in a similar manner. The compress of linen is moistened with water at 80° to 85° F. With robust children the water may be at 70° F. The compress is wrung out and applied so that the neck, shoulders, and chest are covered as with a shirt. The flannel is now applied to the outside. The compress is moistened every hour with water at 70° to 85° F. and re-covered with the flannel.

The **cold pack** is not so useful in the treatment of the febrile conditions of childhood. The method is similar to that followed with the adult, with the exception that the sheet is moistened with water at 80° to 85° F. In other cases the patient, after being wrapped in such a sheet, is rubbed by the attendant with ice on the outside of the sheet. The author has had no extensive experience with this method.

The Full Bath.—The full bath, as advocated by Brand, is seldom carried out in the treatment of children. Children struggle against the bath, and if the temperature is too low, they become so depressed that it is difficult to rouse them. I therefore place children with typhoid fever, or pneumonia, or scarlet fever in a bath at 100° to 105° F., and lower the temperature to 80° or 85° F., applying friction to the body constantly. After five or ten minutes the patients are taken out of the bath and rubbed dry. Warm-water bottles are applied to hands and feet.

In conditions of delirium and coma with a high temperature, in which the heart is weak, I have given baths at a temperature of 105° to 108° F. The cases in which these baths are indicated are those in which any application of cold water causes cyanosis and collapse. I have seen infants suffering from bronchopneumonia, with high temperatures, in a condition resembling a rigor after a bath at 85° F. With these infants the warm bath acts as a cardiac stimulant and quiets the nervous symptoms.

Hypodermoclysis.

Hypodermoclysis is the injection into the subcutaneous tissue of either a 0.6 per cent. salt solution or the normal salt solution of Cantani (sodium chloride, 4 parts; sodium carbonate, 3 parts; water, 1000 parts). It is indicated in infants suffering from cholera infantum and in other exhausting states. Monti, who was the first to apply this mode of therapy to the infant, injects 100 to 200 c.c. at a time. Epstein showed that smaller quantities—10 to 40 c.c.—are more beneficial and more quickly absorbed. Experience teaches that large quantities of fluid injected subcutaneously cause extensive blood extravasations in exhausted infants and much subsequent pain. The solutions used should be freshly prepared and sterilized. Welch has reported cases of infection with Bacillus aërogenes capsulatus following hypodermoclysis. I have had one case, although every precaution was taken to avoid infection.

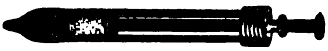
A large antitoxin syringe, holding 20 c.c., is used. It should be carefully sterilized.

From 20 to 30 c.c. of the solution are injected two or three times daily into the subcutaneous tissue of the lumbar region or abdomen. Monti injects into the subcutaneous tissue of the abdomen. Massage should not be performed after injection, as it is very painful and causes hemorrhages. The puncture wound is covered with a piece of sterile gauze. The main point is to inject small quantities of the solution at intervals of from four to six hours, and watch the effect. The action is that of a stimulant to the heart and the processes of resorption. Epstein showed that within a few hours after injection of salt solution the proportion of hæmoglobin and red blood-cells was reduced. As salt solution has a dissolving effect on the red blood-cells, the injection of large quantities of the solution may be harmful.

Syringing of the Nose.

Instruments.—The best form of syringe for this purpose is an olive-tipped glass syringe. Some forms are made with a soft-rubber tip. The tip should be blunt, lest the nares be injured (Fig. 7).

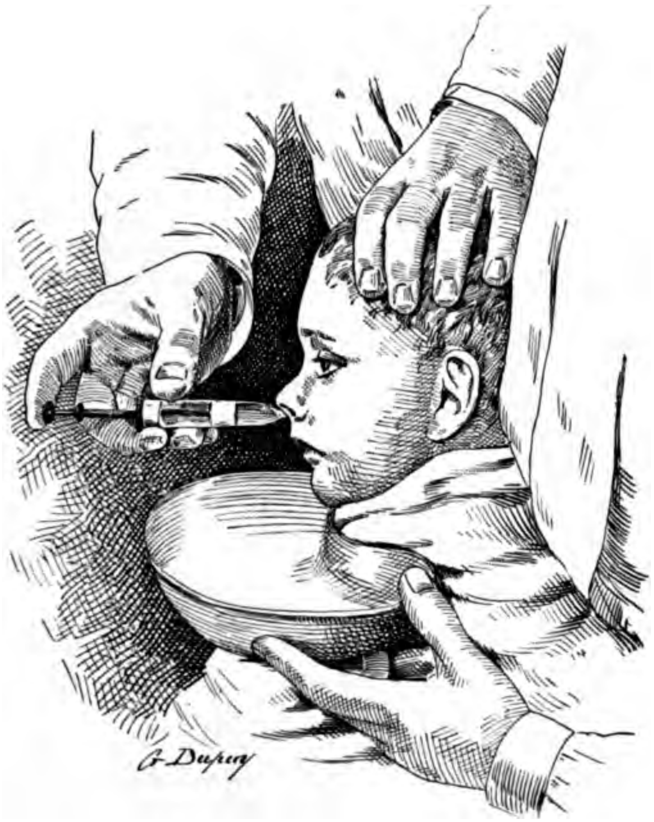
FIG. 7.



Nasal syringe. Correct shape.

The solution used is generally a normal salt solution.

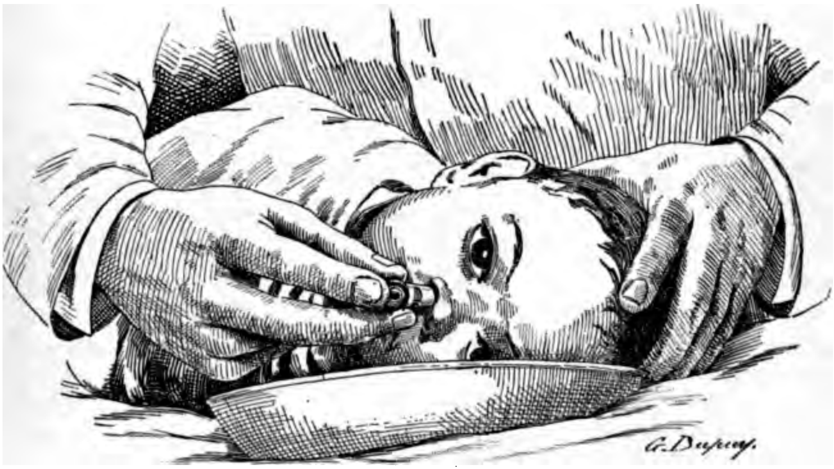
FIG. 8.



Method of syringing the nose in the upright posture.

Method.—The infant or child is wrapped in a sheet or blanket, and held in the lap of a nurse, who holds a pus basin beneath its chin. The operator stands behind the patient. The syringe is held horizontally to the floor of the nares and the solution slowly injected into the nostril (Fig. 8). If successfully performed, the procedure results in the solution's coming out of the other nostril. There is no danger in the manœuvre if carefully carried out. If the infant is too weak, the nares may be syringed with the patient in bed in the recumbent posture. The nurse stands at one side, and the head is placed on the side, the pus basin beneath the nose, as shown in Fig. 9. A rubber fountain-syringe may be used in the same manner.

FIG. 9.



Method of syringing the nose in the recumbent posture.

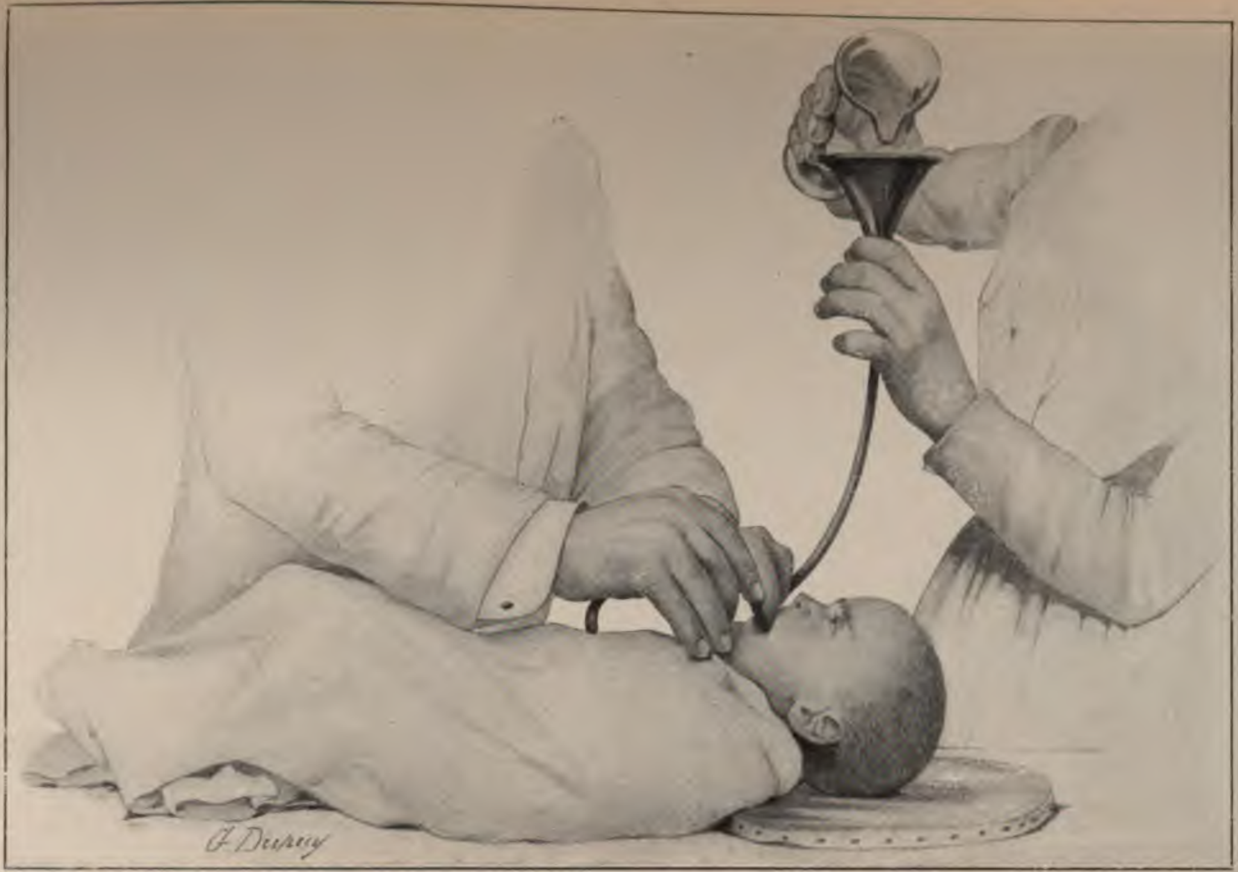
Here also the position of the syringe is horizontal to the floor of the nares. The syringe should be thoroughly boiled before and after using. An old syringe should never be used, no matter how carefully it has been sterilized.

Vapor Spray; Calomel Inhalations in Acute Laryngeal Disease.

With infants and children the spray is not so useful an agent as steam vapor impregnated with balsams or turpentine, and combined at times with inhalations of the fumes of sublimed calomel. The spray cannot, as a rule, be used locally except with the most tractable children. With infants its use is not feasible.

The vapor of steam impregnated with balsams or turpentine is very useful in all forms of acute laryngitis in which there is no bronchitis. I dispense with steam vapor if bronchitis is present. The mode of application in catarrhal or membranous croup is as follows: The crib is covered with a sheet suspended from four

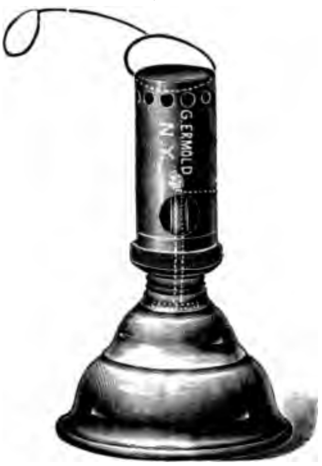
PLATE I.



Method of Washing Out the Stomach in the Recumbent Posture.

upright poles fastened to the corners of the crib. A tent is thus formed. The croup kettle is placed at one side of the crib, in such a manner that the steam vapor escapes into the improvised tent. The vapor is medicated by placing in the kettle a teaspoonful of turpentine or thymol. This will be readily vaporized. No special apparatus has any advantage over the ordinary croup kettle. If calomel sublimations are to be given, they should be combined with the steam vapor. Ten grains of calomel are placed in a spoon held over an ordinary candle, and the fumes led under the tent, the air of which is impregnated with steam vapor. The special devices sold for the sublimation of calomel may be used, but possess no advantage over the method described above (Fig. 10). Calomel sublimations are exceedingly irritating, but they relieve the patient very promptly. They may be continued for forty-eight hours at intervals of two hours, without fear of salivation.

Fig. 10.



Sublimer for calomel inhalation.

Stomach Washing.

One of the most valuable additions to our therapeutic armament within recent years is stomach washing in case of the nursing infant. No improvement has been made upon the method as first proposed by Epstein. The cases in which it is indicated are mentioned in another part of this work. The procedure is easiest of application to nurslings in whom there are no teeth or in whom very few teeth have erupted. With these subjects there is no danger of the catheter's being bitten, and there is no necessity of using a gag. With older children, however, a gag must be used when stomach washing is attempted. The Denhardt gag of the O'Dwyer set of intubating instruments is most suitable for this purpose.

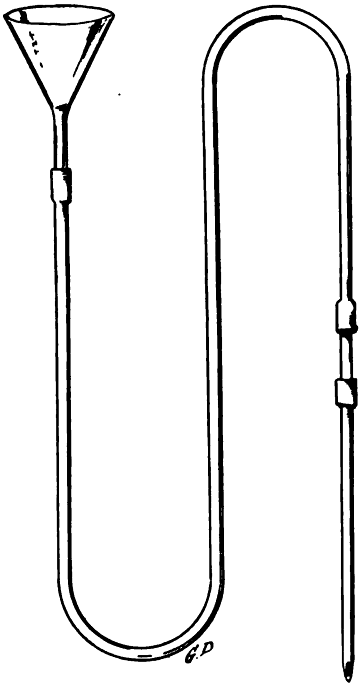
Indications.—Washing out the stomach is principally indicated in the acute gastro-enteritis of the summer months. It is not bottle-fed infants alone that are attacked, but even breast-fed infants may be thus affected. The winter months also furnish their quota of these cases. One vomiting spell, as it is called, does not require attention. If, however, on suspension of the bottle or breast, vomiting continues and becomes uncontrollable, we proceed to stomach irrigation. Another indication is the so-called chronic dyspeptic vomiting. Those who advocate this method of treatment in these cases forget that, above all, the food is at fault, and must be regulated and modified. I do not favor washing the stomach in these cases.

One washing is, as a rule, sufficient. I have rarely had to repeat it. If vomiting persists after the first washing, it is well to look for other conditions than gastro-enteritis, such as intussusception, as the cause of the vomiting.

Acute drug poisoning or ingestion of any irritating fluid is quickly relieved by stomach washing. I have washed out many children who had been given an overdose of paregoric, or who had taken Paris green, turpentine, or other drug. If, as sometimes happens, a child accidentally swallows a caustic alkali, we should not introduce the tube into the œsophagus or stomach.

Method.—A four-ounce funnel, a piece of rubber tubing two and a half feet long, and a No. 14 rubber catheter are the instruments necessary. The rubber tubing is attached to the funnel, and by means of a piece of glass tubing to the catheter, as in Fig. 11. About a quart of normal saline solution is needed. The temperature

FIG. 11.



Apparatus for washing out the stomach.

of the water should be at least 100° F. The operator needs one assistant. The infant is completely undressed, and is then wrapped in a blanket, the diaper having first been applied. The hands are tucked in with safety-pins. The infant having been laid recumbent on a table, the operator, standing on the right, introduces his left index finger into the mouth and depresses the tongue (Plate I.). The catheter, moistened with water, is now introduced and passed backward. With gentle urging the catheter passes easily into the œsophagus. There is no likelihood of the catheter's passing into the larynx and trachea. About six inches of the catheter are introduced. The funnel is depressed and the stomach contents are first allowed to flow out. The funnel is then raised about two feet above the patient, and the assistant slowly pours the saline solution into the funnel, the fluid flowing into the stomach. Before the funnel is completely emptied, it is lowered and the stomach contents siphoned out. This operation is repeated several times, until the water returns quite clear. If during the stomach washing the fluid should be ejected from the stomach in the act of vomiting, it will easily flow out of the mouth if the infant is recumbent. There is not the slightest danger of aspiration of the fluid into the trachea. I think the recumbent position is superior to the sitting posture advocated by some clinicians. A young infant is unable to sit up of its own accord. The introduction of the tube is not so easy for the infant in the sitting posture as in the recumbent position. The tube being introduced, the stomach contents sometimes refuse to flow out because mucus and food particles obstruct the lumen of the catheter. In such cases the catheter is withdrawn, and washed out. The catheter is then pinched with the fingers in such a manner that some of the water or washing solution remains in the catheter. It is then introduced into the stomach. In this way the catheter, being filled with fluid, mucus and food cannot obstruct the lumen of the tube before siphonage is begun. Fluid can then readily be introduced into the stomach. These difficulties occur in cases in which there is a large amount of mucus in the stomach. The finger should always be retained in the mouth. By grasping the catheter with the thumb and index finger of the right hand, prying open the mouth at the same time, we prevent pressure on the catheter during the washing. If the infant has upper and lower incisors, the catheter must be held at one side of the mouth and the mouth kept open by means of the index finger held in the angle of the mouth. The method described above has been followed by me for years. I have never had an accident.

Gavage.

Gavage is a method of forced feeding by means of the stomach-tube. I have not practised this method of feeding infants. Older children suffering from pneumonia or typhoid fever, and delirious or unconscious, have been fed with success by this method in my wards.

The method of procedure is similar to that used in stomach washing. It is best not to introduce the catheter through the nose, but to keep the mouth open with some device. If the catheter is passed through the nose, no food should be introduced into the funnel until we are sure the feeding-tube is in the stomach. With older children a tube passed through the nose may pass into the larynx. If it has done so, a hissing sound will be heard. Aphonia will also be present. In infants and young children the glottis is small, and a full-sized catheter will not readily pass into it. After the tube is in the stomach the prescribed amount of liquid food is introduced and the tube rapidly withdrawn. The feeding may be repeated every four to six hours.

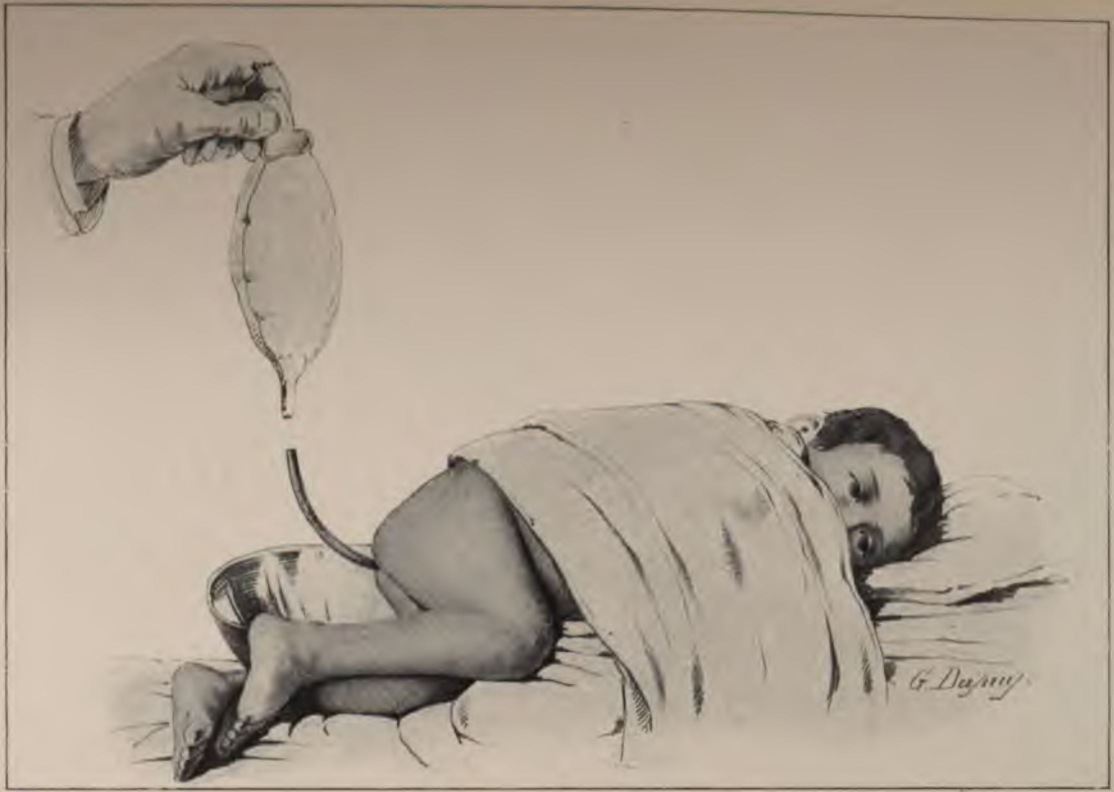
Rectal Enemata; Irrigation; Enteroclysis.

The bulk of an ordinary enema, introduced in order to empty the bowel, should be from 2 to 4 ounces. A Davidson's bulb syringe should not be used. A No. 16 or No. 18 catheter is attached to the nozzle of an ordinary four-ounce hard-rubber syringe. The infant or child is placed on its side, with a rubber sheet under the buttock. The tip of the catheter is oiled and passed well within the anal ring. The catheter is then attached to the nozzle of the syringe containing the fluid to be injected, and the fluid is gently thrown into the rectum. An enema commonly used is soap-water, with the addition of a tablespoonful of castor oil or glycerin.

The high rectal enema, irrigation, or enteroclysis, is given in all forms of summer diarrhœa, dysentery, and in typhoid fever. It is also indicated in cases in which there are symptoms of collapse, in exhausting diseases, in nephritis, and after operations. It is also used to reduce intussusception. In diarrhœa, the object of the high rectal enema is twofold—to clear out the feces from the lower bowel, and to supply fluid to the depleted circulating blood, thereby stimulating the heart. The latter is the main object in practising enteroclysis in states of exhaustion and after operations. In suppression of urine we aim to supply fluid to the kidneys and stimulate the circulation. According to Kemp, the high rectal enema is one of our most useful diuretics.

The solution employed is the Cantani saline solution (sodium carbonate, 3.0; sodium chloride, 4.0; water, 1000). At least a quart is injected. The temperature of the solution for simple washing of the gut, as in diarrhœa, should be that of the body. In nephritis or collapse the temperature should be at least 108° to 110° F. (42.2° to 43.3° C.).

PLATE II.



Method of Giving a Rectal Enema.

The instrument employed may be a bag fountain syringe, of a quart capacity, to which is attached a small calibre soft-rubber rectal tube or a catheter, or the rubber tubing and catheter may be attached to a six-ounce glass funnel.

The patient is completely undressed and laid on a table on the side, with the knees flexed and the buttocks near the edge. A rubber sheet placed underneath the buttocks leads into a pail, so that the returning water will drain off (Plate II.). The buttocks are placed slightly higher than the trunk. The catheter or rectal tube is oiled and introduced two or three inches into the rectum, the water allowed to flow, and the tube passed higher up. Sometimes there is an obstruction to the passage of the tube, and then it is necessary to introduce the finger cautiously into the rectum alongside of the tube and guide it past the upper sigmoid ring. The tube may thus be passed from six to eight inches into the gut. It is seldom necessary to introduce it higher, as the water will find its way into the colon. About a pint or more of water is then allowed to flow into the gut. It is not necessary to compress the anus around the catheter to prevent escape of the fluid. Some of the fluid may escape alongside the catheter. In some forms of exhausting diarrhœa a portion of the saline solution should be left in the gut after it has been well irrigated, in order to stimulate the heart and supply fluid to the circulation. Two irrigations may be necessary in the twenty-four hours, rarely more. In typhoid fever one low irrigation is given daily. In some subjects, if the irrigations are continued too long, hyperæmia of the mucous membrane results. Clinically, this is manifested by a continuance or increase of mucus in the washings, and also by the occasional presence of blood. In such cases the enemata must be suspended. In nephritis complicating scarlet fever, rectal irrigation is one of the recognized methods of stimulating the secretion of the kidney, which result, according to Kemp, begins twenty minutes after the fluid is introduced into the gut. With adults the Kemp tube is used, but with children, who are difficult to keep quiet, continuous irrigation is not feasible. In these cases high enteroclysis is given in the ordinary manner, as much of the solution as possible being retained in the rectum. This procedure may be repeated two or three times daily. In giving ordinary enteroclysis the bag of the fountain syringe or funnel should not be held more than three feet above the body of the patient, lest the pressure be too great. About a pint of fluid at a time is allowed to flow into the gut; the catheter is then disconnected, and the contents of the gut allowed to flow out.

A stimulating enema is given after an operation, or when symptoms of collapse appear in any acute illness. Only small quantities of solution are allowed to flow into the rectum. A formula in use in my wards is the following:

Whiskey	ʒj.
Caffeine	gr. ½.
Tinct. digital.	gtt. ij.
Sol. sodium chloride (0.6 per cent.)	℥j.

Temperature, 102°–105° F.

Nutritive enemata are used when for any reason, such as uncontrollable vomiting, the stomach must be given complete rest. Somatose solution, of one teaspoonful of somatose dissolved in eight ounces of cold water, is given lukewarm ℥ij at a time, every four hours. Or, ext. pancreatis, gr. v; sod. bicarb., gr. ij; water, ℥iv; milk, ℥xvj; with or without the addition of an egg. Give ℥ij or ℥iij. These enemata should be given slowly and high up, and in small quantities at a time.

For constipation the following is excellent:

Olive oil	℥ij.
Glycerin	ʒj.

SECTION II.

INFANT FEEDING.

I. PRINCIPLES UNDERLYING THE PROCESSES OF NUTRITION.

THERE is no division of pediatrics which exceeds in importance that of infant-feeding. In fact the subject of infant-feeding is not only difficult to master, but requires thorough study and experience to carry it to a successful issue. The practitioner, therefore, will find that it is absolutely necessary for him to understand the principles underlying the art of infant-feeding, in order to attain any success in practice in this field. Though great advances have been made in the study of infant-feeding in the past decade, we cannot say that the art of applying certain principles of nutrition to the feeding of infants has attained its highest perfection. We cannot explain why one infant will thrive, whereas another, fed according to the same method, will fail to thrive and lose ground. To a certain extent, therefore, the subject of infant-feeding is still empirical, although it may be said that empiricism is gradually but surely disappearing from this field of pediatrics. It is the exceptional infant which to-day refuses to thrive, and puzzles the most brilliant master of the art, but the vast majority of children can certainly be fed according to principles well established and laid out at the disposal of the general practitioner.

If we study these principles of nutrition closely we shall see that they must to a certain extent conform to what is known to take place not only in the body of the infant, but also in that of the adult. There are certain exceptions which must be made as regards the infant, on account of its rapidly growing organism and the fact that the cells of the body are not only being replaced rapidly, but the tissues at the same time are undergoing rapid increment. As that of the adult, the body of the infant and child is constantly suffering a loss of its principal elements, consisting of water, albumin, fat, and mineral salts. This loss will vary within wide limits, according to the needs of each individual subject. The infant body must take in sufficient nourishment not only to make up for the constant loss and destruction of cell life, but also for the increase and growth of the body and development of various tissues, in this respect differing from the adult. The loss of nitrogenous substances and fat must be made up by equivalents in the food; at the same time in the infant and child enough must be furnished to allow for the rapid increase of weight and the growth of tissue throughout the body.

There are other substances, such as collogen, chondrogen, keratin, mucin, and lecithin, which are needed in the infant's economy as well as in that of the adult, and these are excreted by the infant and child as in the adult. If fat and albumin are taken in sufficient quantity into the system, the loss in these substances is compensated for by the splitting up of the nitrogenous and fatty elements of the food.

It will enlighten the student to familiarize himself with the rôle played by the various food elements in replacing the loss of tissue in the economy. These primary food elements are principally water, mineral salts (inorganic and ash residue), proteids, or albumin, fat, and carbohydrates.

Water.

Water plays by far the principal rôle in the composition of the body. The tissues of the body contain from 66 to 70 per cent. of water in the newborn infant and child, as compared with 64 per cent. in the adult. It exists in this high percentage in most of the organs of the body, with the exception of the bones, cartilage, teeth, and fatty tissue. The remaining organs, if these be excluded, will contain 78 per cent. of water. Water is not only essential to the adult body, but is a very important element of nutrition in infants. We see this exemplified in disease, especially when the drain on the system is great and the loss of fluids of the body is considerable, as in cholera infantum, or in intestinal disease, acute and chronic. Infants show the drain of water from the economy very rapidly, and our treatment in disease is directed in a great many instances to supplying the loss of water caused by the diseased condition. The circulation of the blood and lymph depends on the presence of a fixed percentage of water; in the former case 78 per cent., in the latter 96 per cent. of these tissues is composed of water. Digestion, both in the adult and the child, must have for its successful completion a certain amount of watery element. Muscular and nerve force are greatly dependent on water and are regulated by it.

The body excretes water through the urine, the fæces, the lungs, the skin, and the amount excreted varies widely, not only in the adult, but in the infant and child.

It is not our aim here to enter into any specific details of the rôle played by water in the economy, but from what has been said it can be seen that inasmuch as fully 86 per cent. of the breast-fed infant's food consists of water, nature has put great store by this element of foodstuff which is taken into the infant's body daily. Moreover, water given in disease will sometimes maintain life, but it cannot maintain the proper nutrition of the body without the addition of other elements of food. This is seen in the treatment of gastro-enteritis. We may tide over a critical period in the disease by the administration of water exclusively, without endangering life through starvation. During this period, however, the nitrogenous waste of the body is not replaced by any equivalent article of food, and though we may continue on a water diet for a little while, it becomes imperative after a time to add other substances to the food.

Mineral Salts.

Mineral salts exist in most of the tissues of the body and in all organized tissue which, when burnt, leaves an ash residue. Sodium, potassium, lime, magnesium, and phosphorus, with a trace of iron, are the principal mineral substances found in the body. Just as water is necessary to the maintenance of the nutrition of the body, so are the mineral salts. The actual growth of the child in the first six months amounts to 150 to 300 grammes; in the following six months, 100 to 200 grammes per week. In the second year the body-weight is increased by 50 to 100 grammes per week, and from this time on the increase declines. The skeleton in the first year increases fully 2.2 pounds, or one kilo, in weight, and the earthy phosphates being an important element in the composition of the bones, 3.5 grammes of phosphate of calcium are used every week during the first year by the skeleton. This great demand of the skeleton for lime salts is met by the food of the infant—the milk—much better and in a more assimilable state than by any food taken by the adult subject. The muscles also need a certain amount of lime salts, and a dearth of mineral salts becomes evident much more quickly in the infant and child than it does in the adult. We see this exemplified in artificially fed infants, whose food (cows' milk) is not as well assimilated as is the mother's milk by the naturally fed infant. Whereas 800 c.c. of mother's milk contain 1.2 grammes of potassium phosphate, 0.2 grammes of lime phosphate, 0.6 grammes of sodium chlorid, and 2.5 milligrammes of iron, and these are completely assimilated by the infant, the same salts in cows' milk are excreted to a great extent by the intestine (Bunge), and for this reason, in part, rachitis and disturbances of nutrition of the bones are very common in artificially fed infants.

Proteids.

Next to water, according to Munk, the most important constituents of the body are the proteids; they make up 10 per cent. of the tissues. The proteids in the food not only replace the general nitrogenous loss of cell tissue in the body, but with other substances, the so-called proteid-saving elements of the food, such as fat, add to the general nitrogenous store in the body. Nitrogenous cell waste can be replaced only by the proteids of the food. Growth of body is accomplished by the proper supply of albumin in the food. Whereas other substances, such as fat, added to the albuminous substances of the food may *replace* nitrogenous waste in the body; increase of weight or growth can be accomplished only by the proteid elements of the food. The bone tissue, cartilage, tendon, connective tissue, need proteids also, as has been stated above, to replace the waste and accomplish the growth of these tissues. The breast-fed infant obtains in its food a casein and also, in small quantities, lactalbumin. From these the body forms not only the nitrogenous cell elements, but mucin, chondrin, glutin, elastin, keratin, which are derivatives of albumin, and whose mode of formation is still obscure (Munk).

Fats.

Animal fats are composed of varying proportions of olein, palmitin, and stearin. Their presence in the body varies, within certain limits, from 9 to 23 per cent. of the body-weight. Fat is found in the body in the form of fat-deposits. It is deposited underneath the skin, in the muscle, in the nerve tissue, around the various organs of the body. It plays an important rôle in the maintenance of the warmth of the body and exerts a non-conducting rôle, preventing radiation. As a food it cannot replace the proteids. Fat combined with proteid substances in the food may, however, act as a nitrogenous-saving substance. Thus, in muscular work the body needs a great amount of fat. If combined with the proteids, nitrogenous waste is saved and fat is burnt up in doing the muscular work, and it may even, if taken in sufficient quantities, cause an accumulation of fat in the body. To cause growth in nitrogenous tissue, however, the presence of a sufficient amount of proteid in the food is absolutely necessary. Thus, while fat and albumin may replace waste caused by muscular action, both in the fatty and nitrogenous tissues of the body, fat cannot add to the nitrogenous growth of cell tissue. The infant and child obtain the fatty elements of the food in the milk. Whereas 97.5 per cent. of the fat in mother's milk is assimilated, only 93.5 per cent. of the fat of the cows' milk is assimilated by the infant. The artificially fed infant, therefore, is deprived of an important food element to the extent indicated, and in many cases assimilation of fats in the artificially fed infant is even much more imperfect in practice than is indicated by the percentage named. For we note that in some infants, if the fat in the cows' milk is increased beyond a certain percentage, certain symptoms of intestinal indigestion manifest themselves in a so-called fat diarrhœa, and it is a common observation that such infants must be fed on a limited amount of fat because of the difficulty of assimilation of fat of cows' milk.

Carbohydrates.

According to Munk, carbohydrates exist in various tissues of the body, most abundantly in the liver, in the form of glycogen and grape sugar; in the human milk, in the form of milk sugar, 3½ to 9 per cent., in the muscles, in the form of glycogen, 0.3 to 0.9 per cent., with some grape sugar. The blood and lymph contain a small quantity of grape sugar (0.1–0.15 per cent.). We find glycogen in all growing tissues, and the formation of glycogen seems to be a function of the young cell.

The infant obtains its carbohydrates for the most part from the milk, where they exist in the form of milk sugar. Milk sugar as contained both in human and in cows' milk is assimilated by the infant completely, so that in this respect the infant is not deprived in artificial feeding of any food element.

Carbohydrates play much the same rôle in the economy as do the fats in saving nitrogenous waste. Whereas we can make up to a certain extent nitrogenous waste by the addition of fats and carbohydrates to the food, the nitrogenous substances of the body themselves can be reproduced only by nitrogenous proteid substances. It is self-evident, therefore, that in infant-feeding, though we may produce fat by carbohydrates, saving to a certain extent nitrogenous waste, we cannot do this for any length of time without producing an actual proteid starvation unless we supply with the carbohydrates and the fat a certain amount of proteids. We see this well illustrated in substitute infant-feeding in cases of difficult proteid digestion. We can aid digestion of the proteids by the addition of carbohydrates. We can even cause the formation and deposit of fat to a great extent by the addition to the food of carbohydrates. We can save nitrogenous cell waste by the addition of carbohydrates to the food. If we continue this mode of feeding for any length of time we can see clinically the effects of the dearth of proteids on the economy. The infants after a period of time do not increase in weight, the tissues of the body suffer in nutrition, and anæmia appears. We then must supply with the carbohydrates an increased amount of proteids.

II. METABOLISM IN THE NURSING INFANT.

The principles of metabolism and nutrition which have been established in the adult apply in a general way to the nursing infant. In the adult the food supplies the waste and maintains body-heat and energy, but in the infant it must also furnish, in addition to these, the material for body-growth. The main physiological characteristic, therefore, of infancy and childhood is that it is a period of growth, and the younger the infant the greater the growth.

Milk, the food of the breast-fed infant, contains all the necessary food elements to maintain nutrition, produce energy, warmth, and to aid in cell-growth. In considering metabolic processes in the infant we express the energy and warmth-producing equivalents of the food introduced into the body by the term calories. A calorie is the heat produced by raising 1 kilogram of water, 1° C., and is the unit of heat. In the infant there is a deficit, as in the adult, of 10 per cent. between the raw calories (food) introduced into the body and the actual number of calories produced. In other words, all the food is not absorbed. We do not know as yet how much to allow in estimating the number of calorie equivalents for the excreta, urea, carbonic-acid gas, and water. With the above defects yet to be elucidated by further investigations, we can present the following facts:

A breast-fed infant, three months of age, weighs 5 kilos, takes 800 c.c. of breast milk in the twenty-four hours, and increases 0.25 to 0.35 grammes a day. A litre of human milk contains: casein 16 grammes, fat 35 grammes, milk sugar 65 grammes. The adult, on the other hand, takes daily 1.7 of proteids, 0.85 of fat, 7.5 of carbohydrates per kilo of body-weight. The nursing infant, therefore, takes per kilo of body-weight twice as much proteids and three times as much fat as the adult, the milk sugar being converted into fat values. In the adult the ratio of proteid to other food substances is as 1 to 5 in the food; whereas in the infant taking human milk the ratio is as 1 to 6, and with cows' milk, 1 to 3.

According to Rubner, the caloric value of 1 gramme of proteid substance of the milk is 4.4, 1 gramme of milk sugar, 3.9 calories, and 1 gramme of fat, 9.2 calories. One litre of human milk is equal to 650 calories, and 1 litre of cows' milk to 700 calories. An infant three months of age, therefore, drinking 800 grammes of breast milk would take in 500 calories daily, and if it weighed 5 kilogrammes it would be taking 100 calories per kilogramme of body-weight a day. Bonnoit found by experiment that an infant produced 80 calories per kilogramme of body-weight in twenty-four hours, and if we deduct 10 per cent. from the raw caloric equivalent of the food we would have almost as many calories introduced into the body as the body produced.

The need of 100 calories per day remains constant during the first year of life, diminishes slightly in the second year, with the following exceptions: During the first ten days the infant uses up only 40 to 50 calories, and the increase of weight is accomplished mostly by the watery substances of the food. Rubner and Heubner found that of the 100 calories used up daily by the infant, 20 were utilized to supply body-waste and 80 were burned up to produce heat. Therefore the necessary heat-producing calories are much higher in the infant as compared to the adult, as are also the number of calories necessary to increase body-weight. This greater need on the part of the infant is explained by Rubner by the fact that in proportion to their body-weight, infants present a greater surface area than do adults, and therefore lose much more heat in a given time than does the adult subject. Therefore the extent of loss of heat is dependent on the extent of surface exposed, and allowing for this and not calculating the needs of the the organism by weight, we find that both the child and the adult need the same number of calories.

The following shows the number of calories produced by the various constituents of the food in the adult and in the infant.

Of 100 calories in the food taken in by the adult, proteids produce 19, fats 30, carbohydrates 51. Of 100 calories in the milk taken by the infant, proteids produce 18, fat 53, carbohydrates 29. In the infant, therefore, the fat is the chief heat producer. It is also nitrogen-saving, inasmuch as the latter is used for cell-growth.

After the first year growth is not so active and less fat is needed, and this constituent is replaced by the carbohydrates. The following table illustrates this :

Age.	Weight-kilogrammes.	Proteids.	Fats.	Carbohydrates.
3 days	3.0	2.4	2.8	2.9
6 “	3.2	3.7	4.3	4.4
4 months	6.0	3.8	4.5	4.6
1½ years	9.0	4.4	4.0	8.9
2½ “	10.0	3.6	2.7	15.0
11 “	23.4	2.8	2.0	11.4
Adult	70.0	1.7	0.85	7.5

Mineral Salts.

The infant in its milk takes more mineral salts into the body than the adult, kilo for kilo of body-weight. They are utilized in the growth of the infant.

Excreta.

Much is to be learned as to how much should be allowed to the excreta in calculating the necessary calories used up by the infant organism. By the excreta we mean urea, water, and carbonic acid gas. Rubner and Heubner have shown that an infant in the first six months excretes less urea than the adult. In the second half year the infant excretes more urea than the adult, and this increases until the tenth year. In proportion to its weight the infant takes more nitrogenous substance into the body than it excretes in the form of urea.

During the first six months, the growth of the infant being most active, this is most markedly so, and the nitrogen is retained to a greater extent in the system during the first six months of infancy. Michael has found that the nitrogen excreted in the fæces and urine and the proteids of the food retained in the body were one-fourth of the whole increase of weight in the newborn infant.

Water.

Rubner and Heubner found that of 530 grammes of water taken by the ten-weeks-old child into the body, 505.5 grammes were excreted, and of this quantity more than half was excreted in the form of urine.

Carbonic Acid Gas (CO_2.)

Voit, Pettenkofer, Förster, and Mensi have shown that from birth to the tenth year of life the child excretes one and a half to two and a half times as much carbonic acid gas as the adult, and this is practically furnished by the fats. Rubner, Heubner, and Bendix, however, have shown that a breast-fed infant weighing 5 kilos (11 pounds) exhales per square metre of *body-surface* less CO_2 than the adult.

Munk thinks that the proteids are utilized in the organism to form carbonic acid gas. The principal facts, therefore, adduced in regard to the breast-fed infant in connection with metabolism are that the infant in the course of the first six months needs for the production of warmth, potential energy, and increase of weight 100 calories per kilo of body-weight. Eighty of these calories are utilized for warmth and energy and 20 for increase of cell-growth. If, therefore, an infant takes only 80 calories into its body, its weight will remain stationary. If it takes less, it will have to utilize its own tissues in order to live, and emaciation will result.

Metabolism in the Bottle-fed Infant.

What has been said of the nursling at the breast applies in a general way to the bottle-fed infant, with the exception that Rubner and Heubner have shown that an artificially fed infant needs 120 calories instead of 100 per kilo of body-weight to maintain warmth, energy, and increase in weight. They explain the need of the additional 20 calories taken into its body by the bottle-fed infant by the necessity of extra work on the part of the intestine in digesting cows' milk. It is of interest that the infant, notwithstanding the fact that cows' milk is so entirely different in its composition from human milk, can utilize this food in the production of caloric energy, as above stated. With this we must consider that the utilization by the infant of cows' milk is not perfect, for we have the following differences between the breast- and bottle-fed infant, which are apparent on the surface. The increase of weight is irregular in the bottle-fed infant as compared to the regular increase in the breast-fed infant. The daily fluctuations of temperature in the bottle-fed infant are irregular as compared to the fluctuations in the breast-fed infant. The bottle-fed infant, as a rule, is an anæmic child; the breast-fed infant the contrary. The bottle-fed infant is rachitic even from birth. It is more susceptible to infection, less resistant to the inroads of disease. It is deprived, notwithstanding its utilization of the calories in cows' milk, of the enzymes and alexins present in the human milk. Therefore the metabolic processes in the infant fed upon the bottle and those on the breast must necessarily differ, and in this respect our scientific data are still incomplete. We cannot replace human milk completely by any form of animal milk known.

III. THE FOOD OF THE INFANT.

In considering the subject of infant-feeding we distinguish between the infants fed at the breast by the natural method and those fed with some substitute for the breast, such as cows' milk or infant foods, or dilutions of the same.

In considering the subject of infant-feeding we will study the food itself, and take up first in order the breast milk.

Human-breast Milk.

Colostrum.—From the third or fourth month of pregnancy the human breast begins to show signs of functionating and secretes a yellowish-white, thick, sticky substance called colostrum. As the period of pregnancy approaches the seventh month the secretion of colostrum becomes more active, and its physical properties are those of a thin, grayish-yellow fluid which exudes from the breast-nipple under slight pressure.

Physical Properties.—Colostrum differs from the normal milk secretion in being of a light-yellowish or grayish-yellow color. It is markedly alkaline in reaction. It is rich in fats and proteids, poor in casein, in that the albumin exists in relatively greater quantity. The composition of colostrum varies from time to time until the period approaches when it is replaced gradually by the normal milk secretion. This occurs about twelve days after birth in a normally functionating breast. At this time colostrum, as such, should have disappeared.

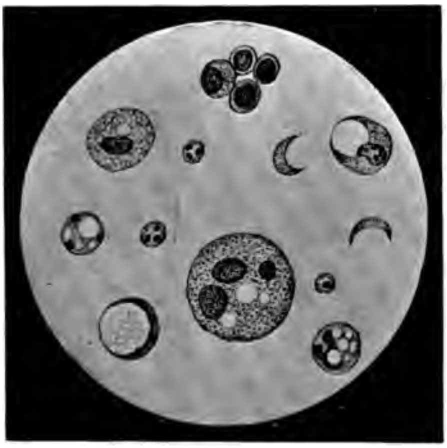
The average composition of colostrum, according to Camerer and Söldner, is as follows:

Water	86.70
Proteids	3.07
Fat	3.34
Milk sugar	5.27
Ash	0.40

It has a specific gravity of 1.040 to 1.060. Microscopically colostrum, in addition to fat-globules, leucocytes, pavement epithelium, granules of casein and phosphates, contains the so-called colostrum corpuscle and the crescent-shaped bodies of Lourié. The fat-globules have similar physical properties to the fat-globules of the milk, and, as in human milk, they are found associated with the cresent-shaped bodies of Lourié, to be described (Fig. 12).

The colostrum corpuscle is a spherical body four or five times larger than the milk-globule, and measuring 13μ to 40μ in diameter. It contains fat in the granular and globular state. The colostrum corpuscle is looked upon as a degenerated leucocyte by some (Czerny). The coloring-matter of colostrum is contained in the colostrum corpuscle. These colostrum corpuscles are the distinguishing feature of colostrum as compared to milk, and so long as they are present in the milk to any appreciable extent the milk cannot be considered as fit, in every sense, for continued infant-feeding. If lactation, for one reason or another, is interrupted, the colostrum corpuscles reappear in the milk. When lactation is again established these corpuscles disappear from the secretion. Should colostrum persist for too long a period in the breast, the infant, as a rule, does not thrive. It can thus be seen that from the twelfth day or thereabout after delivery of the infant, the milk which takes the place of the so-called colostrum must contain either no corpuscles at all or in a vanishing quantity.

FIG. 12.



Colostrum corpuscles and crescents of Lourié. (Marfan.)

In addition to the colostrum corpuscle, colostrum contains an interesting cresent-shaped body, described in connection with human milk, which is seen adherent to the external border of the fat-globule. Some of these colostrum crescents may present an intimation of a nucleus. They have been described by Lourié, and can be seen by extracting the fat from the colostrum and staining with methylene blue or thionine.

Milk.—Milk may appear in the breasts the fifth, sixth or tenth day after delivery. In exceptional cases I have seen the milk delayed as late as the third week; or it may diminish after having appeared and then increase after a few weeks.

There are very many important facts concerning the chemistry of human milk, a knowledge of which is still lacking. Older analyses of human-breast milk give the gross amount of proteids, and Hoppe-Seyler suggested that the casein of human milk, or for that matter cows' milk, should be determined aside from the total quantity of proteids. Therefore the older analyses which deal with the total amount of proteids under the heading of casein are not as useful to us to-day as the more modern analyses which distinguish between the casein and other proteids in the milk. The great importance of this point will become more apparent when we study the composition of cows' milk and attempt to modify it to conform to the composition of human milk.

Composition.—The composition of breast milk varies not only in different women at various periods of lactation, but in the same women at different times of the day. The result is that various analyses differ with each other in a sense, but at the same time agree within certain limits. The student can appreciate these discrepancies by studying analyses of milk given by a number of authors. Whereas there are differences in proteids; these differences have certain limits and with these we should be well acquainted.

König's analysis, as modified by White and Ladd. gives the following composition of human milk and cows' milk:

	Cow.	Human.
Caseinogen	2.88	0.59
Whey proteids	0.53	1.25
	3.41	1.84

The casein in cows' milk comprises five-sixths of the proteids; in human milk, two-sixths of the total amount. We should bear this important fact in mind in reading the following tables compiled from Camerer and Söldner, showing the composition of human milk:

	Ether ext. fat.	Milk-sugar.	Proteids.
Colostrum	5.0	4.5	3.5
Milk, fifth day	2.3	6.7	1.6
Milk, ninth day	3.4	6.7	1.4
Milk, first month	2.6	7.3	1.1
Second and third months	2.4 to 1.9	7.5	0.9

Backhaus gives the following table of average composition (in 100 parts) of human milk:

Water	88.20
Proteids	0.75 casein. 1.00 albumin (whey proteids).
Fat	3.50
Sugar	6.20
Ash	0.25

On comparing these figures with those of König, White, and Ladd, it will be seen that White and Ladd include all the proteids exclusive of casein under the name of whey proteids. The whey proteids are principally lactalbumin and lactoglobulin.

The above analyses tend to show that one examination of any breast milk will give no definite information as to its constant qualities; it will only tell us the composition of that one specimen of milk. In a general way we can speak of averages, and these we shall try to elucidate under the various headings. In order to appreciate the wide variations in the percentages of the proteids, sugar, and fats present at the different periods of lactation, it is further necessary to study the following analyses of leading authorities:

	Proteids.	Sugar.	Fat.
Pfeiffer	1.049-3.04	4.20- 7.60	0.70-9.00
Johanessen and Wang	0.900-1.30	5.90- 7.80	2.70-4.60
V. and J. Adriance	0.230-2.60	5.35- 7.95	1.31-7.61
Schlossmann	0.560-3.40	5.20-10.90	1.60-9.46

Compared with human milk, the following table of animal milk is instructive (König):

	Human.	Cow.	Goat.	Ass.
Water	89.6	87.7	87.3	89.6
Casein	1.4	3.0	3.0	0.7
Albumin	0.6	0.4	0.5	1.6
Fat	3.1	3.7	3.9	1.6
Sugar	5.0	4.5	4.4	6.0
Ash	0.3	0.7	0.8	0.5

Proteids.—There are four albuminous bodies or proteids in human milk. The most important is the casein, which is in a class by itself. The other group of proteid bodies includes the soluble albumins or whey proteids (lactalbumin), globulin, and opalisin. The casein of human milk comprises two-sixths of the total amount of proteids; whereas in cows' milk it comprises five-sixths of the proteids. This is an exceedingly important distinction between the two milks. The casein is, according to reaction, a different casein from that of the milk of lower animals. Szontagh and Wrobelewski contend that whereas the casein of human milk does not yield pseudonuclein on pepsin digestion, it is not a nucleo-albumin, and hence differs widely from the casein of cows' milk. Human milk, as stated, is not only poorer in casein than cows' milk, but the casein is less in proportionate combination with the remaining proteids and lactalbumin. This in part explains the more flocculent nature of the casein coagulum in human milk.

The casein of human milk is derived from the protoplasm of the cells of the mammary gland. It is set free from the cells of the mammary gland in which the fat is formed. In addition human

PLATE III.

FIG. 1.

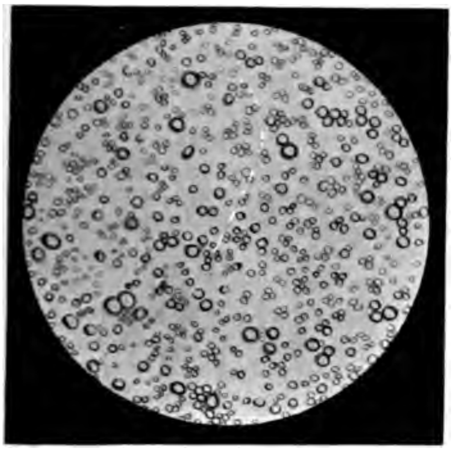


FIG. 2.

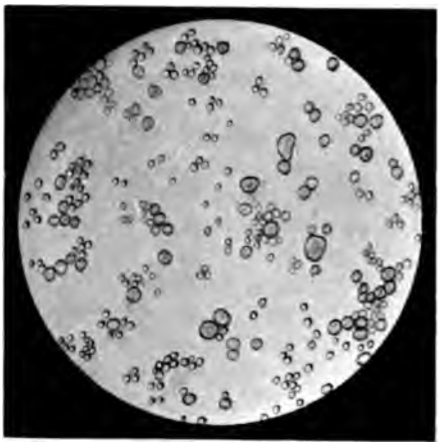


FIG. 3.

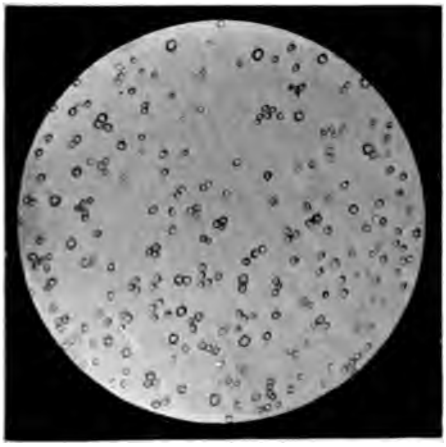
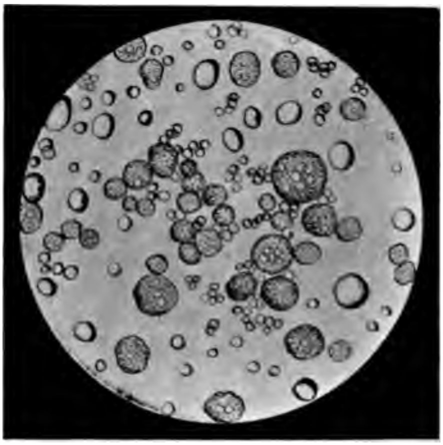


FIG. 4.



Microscopic Appearances of Woman's Milk.

Fig. 1.—Normal milk, showing the preponderance of medium-sized fat-globules.

Fig. 2—Poor milk. Preponderance of large fat-globules and a paucity of fat.

Fig. 3—Poor milk, a paucity of fat and an almost granular state of the fat-globules.

Fig 4—Colostrum of later pregnancy.

Figs. 1, 2 and 3 from Fleischman. Fig. 4 from Marfan.

milk, in the proteids, contains lecithin, 0.58 per cent. (Burrow); iron, 3.52 to 7.21 mg. to the litre (Jolles and Friedjung).

Fats.—The fat of human milk is contained in the so-called fat-globules. On placing a drop of human milk under the microscope, the fat-globule is seen as a highly refracting, spherical body. The globule varies in measurement from 0.001 mm. to 0.02 mm. in diameter (Plate III.), as compared to 0.0016 to 0.01 mm. the size of the fat-globule of cows' milk. It is therefore larger than that of cows' milk. The fat of human milk is a yellowish-white mass, when separated resembling butter, with the specific gravity of 0.966. It melts at 34° C., and is solid at 20.2° C. It contains butyric, caproic, capric, myristic, palmitic, stearic, and oleic acids. It is poor in volatile fatty acids. The non-volatile fatty acids consist of fully 50 per cent. oleic acid, while the palmitic and myristic acids exist in greater quantity than the stearic acid.

In addition to the casein and fat we have the water, 89.6 per cent. Moreover, human milk contains nucleon, 0.124 per cent.; lecithin, 0.58 per cent.; iron, 3.52 to 7.21 mg. to the litre.

If milk is stained with carbol thionine or methylene blue there are seen, as in the colostrum, crescent-shaped bodies which are adherent to the outer border of the fat-globule. They are not nucleolar or remains of nuclei, but are portions of the mammary epithelium which have adhered to the milk-globule at the time of its expulsion from the cell (Lourié).

Mineral Salts in the Milk.—Human milk contains a number of salts, among which are calcium phosphates, potassium, magnesium, iron, alum, calcium and sodium chlorides, sodium carbonate, traces of flourine and silicium. The most important of these salts are the tribasic calcium phosphates, part of which are held in solution, another part exists in a colloid state, the remaining portion being in suspension, and is seen under the microscope as minute dust-like particles in the milk, $\frac{1}{2000}$ mm. in diameter (Duclaux). The tribasic calcium phosphate is insoluble in water, but in the milk is held in solution by the presence of the alkaline citrates.

Salts in the milk.	Human milk.	Cows' milk (Söldner).
Natrium chloride	1.35	0.962
Calcium chloride	0.70	0.870
Calcium phosphate	2.50	1.477
Natrium phosphate	0.40	
Magnesium phosphate	0.50	0.336
Carbonate of soda		
Fluorite calcium	trace.	
Potassium citrate		0.495
Magnesium citrate		0.367
Calcium citrate		2.133
Iron phosphate	0.01	

Reaction.—The reaction of human milk depends on the presence of the contained salts. It is alkaline to litmus and acid to phenolphthalin. The actual quantity of sodium and potassium varies, the sodium being more abundant than the potassium at the beginning of lactation (De Lange). In other words, the reaction of human milk is amphoteric.

Specific Gravity.—The specific gravity ranges from 1.028 to 1.034, being lower in poorly nourished women.

Bacteria in the Breast Milk.—A woman in good health will show bacteria in her milk. The bacteria are found in the galactiferous ducts of the breast nipple. On the expression of the first few drops of milk, therefore, and the washing out of these ducts, it will be found that the after-coming milk is sterile. These bacteria belong principally to the Staphylococcus albus class. The Staphylococcus pyogenes aureus and some few streptococci have been found by Kohn and Neuman. These bacteria have no ill-effect on the infant, and the attempt to trace dyspeptic disturbances to them has been rather far-fetched.

Enzymes and Alexins of Human Milk.—According to the latest investigations, human milk contains certain derivatives of the living cell, which exist in the proteid elements of the milk. Not much is known about them as yet, but their presence proves beyond a doubt that human milk is a substance essentially different from the milk of other animals. Moreover, their presence in the milk and the presence of other substances in animal milk distinguish milk as a living product and not a dead substance.

The enzymes are the soluble ferments in human milk, the most important of which is the so-called amylase, first described by Bechamp and subsequently by Moro. It is capable of converting starch into sugar in the same manner as does the secretion of the parotid gland; in other words, it exerts a diastatic action on starch. Amylase is not found either in cows' or sheep's milk. It is destroyed by heat, and human milk heated above a certain temperature loses its amylolytic properties. This ferment is present, though to a much less degree, in dogs' and asses' milk. It is supposed to be derived from and is a product of the glandular tissue of the mammary gland, and is not primarily present in the blood. The reason of its presence in human milk is not quite understood, inasmuch as the infant reared exclusively on breast milk does not receive any starchy substances with its food.

Marfan isolated another ferment in the milk, called lipase, which is capable of splitting monobutyrin into butyric acid and glycerin. This lipase is present to a slight extent in cows' milk. Human milk contains also a ferment capable of splitting salol into phenol and salicylic acid, and a substance capable of coagulating fibrin, inasmuch as a minute quantity of human milk added to hydrocele fluid causes its immediate coagulation (Moro and Hamburger). This substance is not present in cows' or goats' milk. Moreover, human milk, as also the milk of animals, possesses certain vital specific properties. Bordet, Moro, and others have shown that if human milk, cows', goats' or any other animal milk be injected into the peritoneal cavity of an animal, the serum of that animal in very high dilutions is capable of coagulating the milk of the animal whose milk was injected into its body. This last specific property of the milk of all animals proves beyond question that milk possesses vital properties hitherto not attributed to it.

Human milk contains so-called alexins—that is, bactericidal and globulicidal substances—and Moro has shown that the serum of the blood of the breast-fed infant is more bactericidal than the serum of the blood of the infant fed upon cows' milk.

Amount of Breast Milk Consumed by the Infant in Twenty-four Hours.—Camerer has collated and analyzed the results obtained by Ahlfeld, Pfeiffer, Weigelin, and Hähner as to the quantity of breast milk taken daily by an infant. These figures were obtained by weighing an infant from the earliest period before and after nursing the breast. Camerer gives us the following table, the amounts being indicated in cubic centimetres:

Day.								
1st.	2d.	3d.	4th.	5th.	6th.	7th.	10th.	14th.
30	130	240	290	330	365	400	450	500

Amount of Milk Taken.

	Middle 2d week.	4th week.	7th week.	10th week.	20th week.
Minimum . . .	210	380	520	600	700
Medium . . .	440	580	770	800	900
Maximum . .	540	810	1040	1170	1150

The amount of breast milk consumed by the infant at each nursing must vary with the frequency with which the infant is placed at the breast. If the infant is placed at the breast five times in twenty-four hours the mean quantity of milk taken at each feeding is quite large as is seen by consulting Ahlfeld's figures. Thus an infant at the breast

1 month old consumes	104 c.cm.
2 months " "	163 "
3 " " "	173 "
4 " " "	212 "
5 " " "	212 "
6 " " "	214 "
7 " " "	217 "

These figures are within the limits of stomach capacities given by Pfaundler and in excess of those of Fleischman, Holt, and Rotch. If the infant nurses at more frequent intervals than Ahlfeld's baby, the quantity of milk ingested at each nursing will be less than the above figures.

It is noteworthy that on the first day of life the infant observed by Camerer nursed three times, and seven times in twenty-four hours from the second to the fourteenth day. Each nursing occupied a mean of about twenty to twenty-five minutes. These data are of value in the artificial feeding of infants.

Czerny shows that, as regards the quantity of breast milk secreted, there are, first, those cases in which the breast milk increases in quantity up to the ninth week and then remains stationary in amount up to the period of weaning; secondly, those breasts in which the amount of milk secreted daily increases from the earliest period steadily to the period of weaning, when the amount secreted is at its highest.

Changes in the Composition of Milk.—Daily Changes.—Milk may vary in composition in the course of twenty-four hours in the same nurse, both in the total amount of proteids and fats, to the extent of 1 per cent. or more. Schlichter has found that the changes occur at various times in the day.

The composition of the milk of the nursing woman at different hours of the day may be seen in the following table by Schlichter:

Nurse A.	Casein.	Fat.	Proteids.	Sugar.
Morning	1.10	0.80	1.69	7.11
Noon	1.10	1.88	2.16	6.92
Night		3.16	1.95	6.83
Nurse B.				
Morning	0.55	3.77	1.19	5.37
Noon	0.77	3.90	1.91	6.15
Night	0.71	3.73	1.26	6.19
Nurse C.				
Morning	0.55	3.61	0.19	6.18
Noon	0.83	4.21	1.08	6.24
Night	0.41	3.60	1.16	6.47

Gregor has shown that the variation in the color and consistence of the stools of infants can be accounted for by the variation in the gross amount of fats in human milk at different times of the day and from day to day.

Influence of Foods on Breast Milk.—A diet rich in nitrogenous substances increases the quantity of the milk and the percentage of fats and proteids. A diet rich in fat may increase the percentage of fat in the milk. On the other hand, it is not always possible to increase the casein in the milk by means of diet if the milk is poor in this constituent (König). Starvation lessens the quantity of the milk and the proportion of the casein to the other proteid bodies in the milk in the same manner as does a poor dietary (Decaisne). If we enlarge the diet we improve the milk. Beer and malt liquors increase the quantity of the milk and the percentage of its fat-constituents (König).

In trying to improve the milk of the human breast we should not resort to too much experimentation, for a good milk will sometimes be made unfit for the infant by placing the nurse or the mother on a diet to which she is unaccustomed. On the other hand, I have seen the milk retain its colostrum characteristics through the fact that the mother did not follow out the dietary to which she was accustomed previous to delivery and did not take her usual exercise. Women accustomed to a wholesome, moderate dietary will, if fed liberally with fats and carbohydrates, secrete a milk rich in fats and poor in proteids; such a milk will at once disagree with the infant (König). If a nurse has been on an insufficient diet, the diet should be increased in a general way. She should have a moderate allowance of meat, partake sparingly or not at all of tea, coffee or beer, and have sufficient exercise. If with these changed conditions the milk does not improve both in quantity or quality we should not hesitate to replace the nurse by another; or if the mother is nursing the infant, to aid the breast with artificial food. This is preferable if the mother is nursing to taking the infant away from the breast.

Drugs and Foreign Substances in the Milk.—Iodine and salicylic acid may pass from the blood into the milk of the human breast. Iodine may even cause iodism in the nursing infant when the nurse is taking any appreciable amount of iodide of potassium (Koplik). Iodine is eliminated in combination with the casein of the milk. As to the appearance of other substances, such as drugs, in the human milk if taken in medicinal doses by the mother or nurse much is to be learned, for very little is actually known. Opium is not eliminated if taken in ordinary doses, though atropine may, if taken by the nurse, be eliminated in the milk and cause dilatation of the pupil in the infant. Alcohol taken in limited amounts, as is customary at the table, is not found as alcohol in breast milk; but if large quantities are taken, from two-tenths to six-tenths of 1 per cent. of alcohol may be found in the milk.

Passage of Bacteria of the Infectious Diseases Into the Breast Milk.—The extent to which bacteria of the various diseases may pass into the milk of the infected woman is still a matter of question. Under the heading, Contra-indications Against Nursing the Infant, this matter has been discussed in part. It has been proved that the toxins, antitoxins, and agglutinins of the infectious diseases, such as typhoid fever and diphtheria, may pass into the milk of the nursing woman suffering from these diseases. The bacillary infection of the milk, however, is quite a different matter, concerning which much is to be learned. In local tuberculous infection of the mammæ it can well be understood that bacilli may gain direct access to the milk through infectious foci of the galactiferous ducts. It is still questionable whether milk from a gland free from local foci, though coming from a woman affected with tuberculosis, may contain tubercle bacilli. In typhoid fever and diphtheria the bacilli may appear in the milk of a woman suffering from severe systemic invasion of the bacilli of either of these diseases, but, as a rule, this is not the case. In pneumonia we can scarcely apply to the human subject the reuslts obtained in the lower animals, for in the former the disease is rarely an invasion of the blood to the extent seen in the lower animals. Thus, cases such as those published by Bozzolo, in which pneumococci were found in the milk of a woman suffering from severe pneumonia and endocarditis, are exceptional.

Toxins, Antitoxins, and Agglutinins.—Tetanus toxins and antitoxins may pass into breast milk, and in certain animals, such as mice, this milk may confer immunity on the nursling (Ehrlich, Brieger).

Diphtheria toxin and antitoxin may be eliminated in the breast milk. Ehrlich and Wassermann found that goats immunized against diphtheria could confer this immunity through the milk. Roux and Martin confirmed this observation in the cow. It has also been proved that the breast milk of women convalescent from typhoid fever possessed agglutinating properties on the Eberth bacillus similar to that of the blood (Achard, Bensaude), and that this agglutinating property could be transferred to the blood of the infant nursing this milk (Landouzy, Griffon, and Casteigne). In passing from the blood into the breast milk the agglutinating substance is much weakened, likewise more so when transferred from the milk to the blood of the nursing infant.

The above facts would seem to indicate that the question as to whether the breast milk of a mother or nurse suffering from any disease is fit for the nursling is not an indifferent one. The passage of toxins, antitoxins, and agglutinins into the milk should, with reservations mentioned elsewhere, forbid the use of any breast milk coming from a mother or nurse the victim of active acute or chronic disease.

Menstruation.—The effect of menstruation on breast milk is still a matter of much discussion. Some investigators, such as Rotch, think they have found some variations at this period, not only in the percentage of the fat, but in the proteids, from that which existed before menstruation. Other authors think the greatest variations will be found in the fats (Bendix). I am inclined, however, from my own experience to believe that variations in breast milk during menstruation are exceptional, for the great majority of infants do not show at this time any disturbances of the functions of the gut. An exception to this may probably be the first menstruation of lactation. Infants at this time may have green movements and slight colicky pains which persist until menstruation is established in the mother, when all functional disturbances of the gut disappear and the children do not seem to be disturbed by the recurrence of the function. In fact, if we study the tables of the analyses made before and during menstruation and subsequent to this period we shall see that the variations are no greater than those which occur from day to day when menstruation is absent.

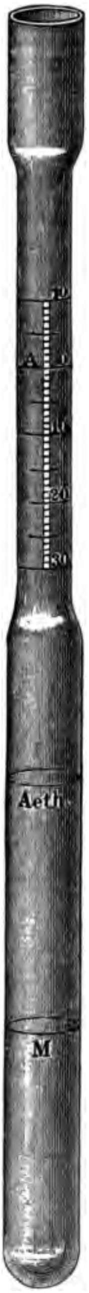
Pregnancy.—The question is frequently asked, "Has pregnancy any effect on the quality or quantity of the milk, and may an infant nurse the breast of a pregnant woman?" Having conducted a very large dispensary class in diseases of infancy and childhood for fifteen years, I not infrequently saw infants nursed at the breast of pregnant mothers. Such infants did not seem to suffer; some of them, in fact, being beautiful babies. Examination of these mothers showed them to be pregnant from four to six months. The milk secretion was not markedly changed in amount. This corresponds to what has been established by Poirier, who found that of 100 pregnant mothers who nursed their infants, 72 infants showed no change in their general well-being, while 20 showed disturbances necessitating immediate weaning. Eight infants showed slight intestinal disturbances. The question may be justly asked whether a like proportion of cases might not be met with necessitating weaning among mothers not pregnant and nursing their babies. Pregnancy may diminish the amount of milk, but in the majority of cases no change occurs. It is just, however, unless extraordinary indications to the contrary exist, that a mother should not be asked to nurse her baby while pregnant with another. Such an infant should be weaned from the breast.

Methods of Analysis of Human Milk.

In the section treating of the examination of breast milk it was shown that with experience it is possible to decide in a general way as to the quality of the milk without chemical analysis. Emergencies, however, arise which may necessitate more careful examination of the milk in order to account for some disturbing symptom in the infant. After thriving for a few weeks the infant may, without apparent cause, cease to gain in weight, or the movements may be abnormal, or there may be colic. Under these conditions it is certainly an advantage to be able to determine the composition of the milk, since a chemist is not always at hand. Conrad, a physician in Bern, has devised some instruments which are easily manipulated and are within the reach of every physician. His article, published in 1880, is still unsurpassed in clearness of detail. The milk to be used in all analyses is that obtained in the mid-period of nursing.

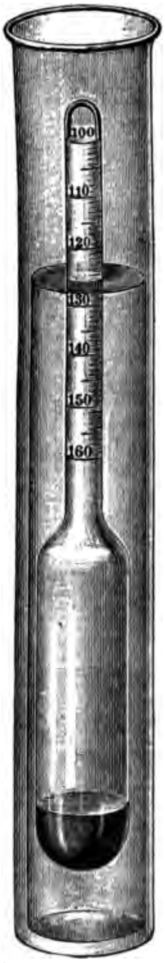
Specific Gravity.—To ascertain the specific gravity, Conrad reduced the size of Quevenne's lactodensimeter so that it could

FIG. 13.



Conrad's lactobutyrometer.

FIG. 14.



Conrad's lactodensimeter.

be utilized for taking the specific gravity of small quantities of mother's milk (Fig. 14). The specific gravity is taken at 15° C. The scale runs from 1020 to 1050.

Fat.—Conrad estimated the fat by first calculating the cream layer. This he determined by means of a graduated glass cylinder devised by Bouchardat, Quevenne, and Chevalier. This cylinder he reduced in size. The method is so unreliable that it is merely mentioned in passing.

Of greater reliability is the Marchand tube, reduced in size by Conrad. The set consists of two of these tubes. Each tube analyzes 5 c.c. of milk (Fig. 13).

Five c.c. of milk are poured into the tube, and then 5 c.c of ether. These are well shaken after a drop of officinal caustic soda solution has been added. Absolute alcohol is then added up to the A mark. The whole is again shaken and placed in water at 35° to 40° C. for ten or fifteen minutes. The fat separates above, and is read off. A percentage table accompanies the instrument. This instrument is not accurate. There is a variation of from 0.2 to 0.5 per cent. or more. Two analyses are made at the same time for the sake of accuracy; hence the two tubes.

Lewi's Method.—More accurate than Conrad's is the method worked out in my clinic by Lewi. This is really an adaptation to breast milk of the Babcock sulphuric acid method, as modified by Leffman and Beam.

The apparatus needed comprises a reduced Babcock bottle, a pipette for measuring the milk and acid, and a smaller 1 c.c. pipette accurately divided into cubic millimetres (see Figs. 15, 16, 17).

METHOD.—Fill the pipette to the meniscus (this represents 2.92 c.c. of mother's milk) and introduce this carefully into the body of the bottle, so that the long thin pipette comes down into the body of the bottle. The pipette is cleansed, and refilled to the meniscus with chemically pure sulphuric acid; the pipette is introduced as before. This precaution is taken in inserting the pipette so that at this stage no ebullition shall occur in the neck of the bottle, and so invalidate the result. Next, fill the 1 c.c. pipette up to the sixth marking with a mixture of equal parts of fusil oil and concentrated hydrochloric acid; add this to the milk and sulphuric acid and fill the bottle with equal parts of sulphuric acid and water. The bottle is placed in an aluminum receiver and adjusted to the centrifuge. The specimens are revolved one and a half to two minutes, and the reading is then taken. With the new high-gear machine of Richards & Co. fifteen revolutions of the handle per minute suffice, each turn of the handle corresponding to one hundred and thirty revolutions of the bottle.

This method, if carefully carrried out, gives very little error, and is practically equal to the Soxhlet

FIG. 15.

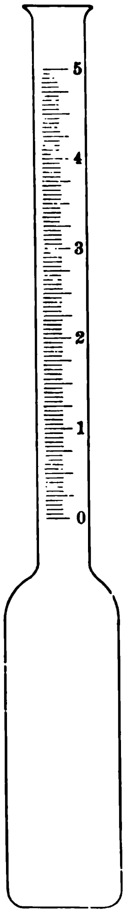


FIG. 16.

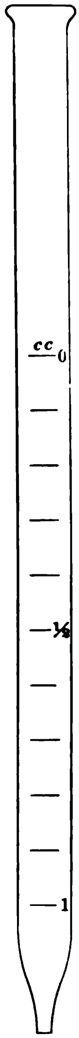
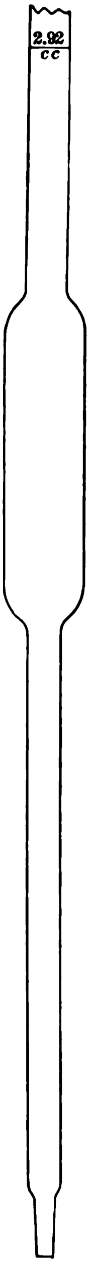


FIG. 17.



Instruments employed in the estimation of fat in milk. Lewi's method.

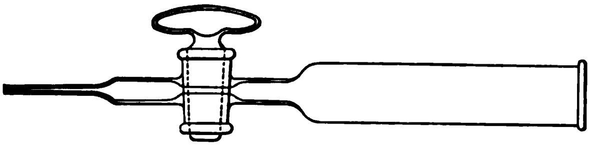
quantitative fat estimation. It can be applied to cows' as well as to human milk.

The following table shows the error in the various methods as compared with accurate chemical determination :

	Soxhlet (chemical).	Reduced centrifuge.	Marchand.	Feser.
Specimen I.	4.4 per cent.	4.4 per cent.	3.48 per cent.	5.00 per cent.
" II. . . .	2.4 "	2.3 "	2.56 "	2.37 "
" III. . .	1.1 "	1.1 "	1.44 "	1.25 "
" IV. . .	3.9 "	3.8 "	3.17 "	3.25 "
" VI. . .	4.6 "	4.7 "	2.35 "	3.80 "
" VII. . .	2.3 "	2.3 "	3.99 "	2.20 "
" VII. . .	4.4 "	4.2 "	3.68 "	4.20 "
" VIII. .	4.7 "	4.6 "		3.60 "

The Proteids.—To possess clinical value in the determination of the proteids, a method must differentiate between the amount of casein and that of the other proteids, such as lactalbumin and lactoglobulin. This is possible only by careful and exhaustive quantitative chemical analyses. The methods at our disposal which are practicable in the physician's office determine only the gross proteids. The gross proteids may be normal in amount, and the casein or caseinogen be deficient. Such milk would not be nutritious. This was demonstrated years ago in sick and starving women (Decaisne).

FIG. 18.



Milk burette of Woodward.

The following is the method of Woodward for determining the total proteids: Two "milk burettes" (Fig. 18), each containing 5 c.c. of milk, are allowed to stand overnight in a warm place (100° F., 38° C.). They are then cooled. The milk is drawn off into two Esbach's tubes, and 10 c.c. of the Esbach solution added. The tubes are then shaken, put into a centrifuge, and rotated until the reading is constant. This method was perfected in the Pepper Laboratory, Philadelphia. The author has utilized this method, and found it satisfactory.

Cows' Milk.

Composition.—Of 700 analyses, König gives the following as the average composition of cows' milk for 100 parts: Water, 87.2; casein, 2.88; albumin (lactalbumin), 0.51; fat, 3.68; sugar, 4.90. Cows' milk has a specific gravity of from 1.028 to 1.034. It is amphoteric in reaction, but is relatively more acid than human milk. Fresh cows' milk does not coagulate on boiling, but heat causes a skin of casein and lime salts to form on the surface of the milk. If allowed to stand at the temperature of the room, lactic acid is formed in cows' milk as a result of bacterial growth and splitting of the milk sugar and coagulation or curdling of the casein occurs when the milk is heated; after a while, an excess of acid being formed, spontaneous separation of the casein will occur.

Fat.—Fat is contained in cows' milk, as in human milk, in the form of fat-globules, which are held in suspension in the serous part of the milk by an envelope of albumin. There is no doubt that the milk-globules contain all the fat of the milk. The fat-globules are smaller than those of human milk. It is uncertain whether the fat-globules contain any protein substances.

Proteids.—**Casein.**—The casein of cows' milk is a nucleo-albumin, contains phosphorus and coagulates when heated, as also by the addition of acids and rennet. The amount of casein in cows' milk is not only relatively but absolutely greater than in human milk; and in describing human milk it was stated that the casein forms five-sixths of the total proteids in the cows' milk; whereas in human milk the casein forms two-sixths of the total proteids. This one fact is of far-reaching importance. Simple dilutions of cows' milk still leave it with a greater proportion of casein, as compared to the other proteids in the milk, than that which exists in human milk. Though we may dilute cows' milk so as to reduce the proteids to the relative proportion in which they exist in human milk, we cannot do this without at the same time reducing its nutritive value; that is, we fail to get the quantity of digestible proteids in the milk, although the proteids may exist in the same proportion in our mixture. In other words, the proteids of the cows' milk are not so completely assimilated by the infant as are those of human milk. Again, the casein of cows' milk is precipitated or coagulates very early with the aid of acid and salts; that of human milk quite late or not at all. In the human stomach, therefore, cows' milk will not take up as much acid of the gastric juice without coagulating as will human milk and the coagula occur in large masses. We can readily see in this another disadvantage in the use of cows' milk as an infant food. Human milk, on the other hand, takes up a large amount of the acid of the gastric juice and coagulates in very fine flocculi. This finer mode of coagulation accounts partly for the more complete assimilation of human milk by the infant. It was formerly thought that the casein of human and cows' milk were chemically identical. Later study, however, shows that the casein of human milk, in contradistinction to that of cows' milk, is not a nucleo-albumin (Szontagh). Human milk is richer in nucleon and lecithin than cows' milk and contains more combined phosphorus than cows' milk in the nucleon. It can be seen from this that the contention of Hoppe-Seyler, Hammarsten, and Wrobelewski, that the two caseins are essentially different, is well founded. Not only is the casein of cows' milk a substance *sui generis*, but its digestion in the intestine of the infant is accomplished with great loss. Paracasein and pseudo-nuclein of cows' milk pass through the gut unabsorbed (Knoepfelmacher). The loss in phosphorus to the infant is sixteen times as great when fed with cows' milk as it is when fed with woman's milk. Moreover, it has been shown that the salts of cows' milk, especially those of lime and potassium, are not well assimilated by the infant gut, fully 34 per cent. of these salts being excreted by the gut; whereas only 10 per cent. of these salts are found in the feces of the infant fed at the breast. These facts are of great importance in comparing the two modes of feeding infants—that of the breast and the bottle. The prevalence of bone disturbances of the severer type in artificially fed infants is thus partly explained, not only by the lack of absorption of an important food element, the phosphorus, but also by the loss of the salts of lime and potassium, these being important to bone nutrition and growth. The increase of weight in artificially fed infants also gives us an insight into the physiological processes in such infants. The quantity of milk, as before stated, necessary to maintain nutrition is greater in the case of the bottle-fed infant than in that fed on the breast. There is always a danger of overfeeding an infant which is bottle-fed. The increase of weight is not as regular in the bottle-fed infant as it is in the breast-fed infant.

The following will show at a glance the differences in the assimilation of the various elements of cows' milk as compared to human milk by the infant gut (Uffelmann):

	Cows' Milk	Human Milk.
Proteids	98.7 per cent.	99.5 per cent.
Fats	93.5 "	97.5 "
Salts	66.2 "	90.0 "
Sugar	100.0 "	100.0 "
Ash	92.0 "	97.0 "

According to Förster, an infant four months of age taking 1215 c.c. of cows' milk excreted three-fourths of the lime salts in the feces.

Bacteria in Cows' Milk.—Pasteurization; Sterilization.—By insisting on strict cleanliness of the cows' udder, the hands of the milkman, and the utensil in which the milk is collected, it is possible to obtain a milk tolerably free from bacteria. In commerce, however, this is manifestly impracticable. Milk collected with the greatest care contains bacteria, and if these appear to the extent of only 9000 to the cubic centimetre at the time of milking, enough will have developed under favorable conditions to cause such an increase within twenty-four hours at an ordinary temperature as to bring this number up to 5,600,000 to the cubic centimetre (Miquel). It is therefore of little value to the practitioner to receive reports as to the number of bacteria to the cubic centimetre when the milk left the dairy. He has no assurance, unless the milk has been kept at a very low temperature, that these bacteria have not increased in number. Soxhlet has shown that in order to inhibit the growth of these bacteria in the milk, it must be kept at a very low temperature, and in summer weather practically in contact with ice.

The most important bacteria found in milk are the Bacterium lactisaërogenes, the Bacillus mesentericus vulgatus (the potato bacillus), and the Bacillus subtilis. Cows' milk may contain also streptococci which come from the udder of the animal, and any pathogenic bacteria, such as the pneumococcus, typhoid bacillus, diphtheria bacillus, the germs of scarlet fever, measles, or tuberculosis, cows' milk being an excellent culture medium for the growth of germs of all infectious disease.

The habitat of the bacteria of cows' milk is first the teat of the udder. The milk ducts in the teats are of considerable size and residual milk decomposes in them. The entrance of bacteria into these ducts, such as the Bacterium lactis aërogenes, the hay bacillus, the potato bacillus (Bacillus mesentericus vulgatus), is favored by the habits of the animal and uncleanliness in the stalls in which the animal is kept. Uncleanly utensils in which the milk is collected are a source of contamination.

Infected Cows' Milk as a Cause of Epidemics.—Typhoid Fever.—Cows' milk is unquestionably an excellent medium for the growth of bacteria and is most readily infected; thus, epidemics of typhoid fever have been traced to infected milk. Such milk becomes infected either in the dairy, where the fever may be prevalent among the dairymen, or through dairy utensils which have been cleansed with infected water.

Dysentery may be caused by drinking infected milk (Klein).

Diphtheria.—The Klebs-Löffler bacillus grows quite well in cows' milk, which may consequently be the means of readily spreading the disease; thus, school epidemics have been traced to infected milk.

Scarlet fever has been conveyed by cows' milk infected by those contaminated with the disease (Kober, Freeman).

Cholera Asiatica may be conveyed through milk diluted with infected water or milk handled by a cholera-infected individual.

Tuberculosis.—It is not the place here to discuss the transmission of tuberculosis to the human subject by means of the milk of a tuberculous cow. This matter is secondary to the more immediate question as to the prevalence of tuberculosis in the infant and child as a result of the ingestion of infected cows' milk. That this mode of acquiring tuberculosis is exceedingly rare will be acceded to by most observers, and published epidemics or isolated cases of tuberculosis in children, caused by infected cows' milk, lack the evidences of absolute certainty as to etiology.

Aside ftom tuberculosis, it is generally granted that suppurative disease of the udder of the cow may cause serious digestive disturbances in the infant by infecting the milk. In fact, certain forms of stomatitis are traced by some (Forcheimer) to such a source.

Milk Acidity.—If milk is not cooled immediately after milking, and kept cool, it soon shows a marked increase in acid reaction. This is due to the growth of the Bacterium lactis aërogenes, which not only turns the milk acid, but in doing so produces toxins which are of considerable danger when introduced into the stomach and gut of the nursing infant. Without entering further into details in this work, we may say that cows' milk intended for infant-feeding should be obtained from a herd of healthy animals, preferably of the Holstein type. Mixed milk is to be preferred to the milk from one cow, for the reason that any infectious element introduced into the milk coming from a large herd of animals is so diluted as to be less dangerous to the individual infant than the milk containing infectious matter coming in a concentrated form from one animal which may not be in good health.

The milk should be carefully collected in utensils which have been thoroughly cleansed and sterilized with steam. The infant should obtain the milk as soon as possible after the milking; certainly within twenty-four hours. Having been modified and put up for the infant's use, the food should be presented to the infant in divided portions, each of which is sufficient for a nursing.

In large cities, where the milk does not come direct from the dairy to the infant, it is still thought advisable to subject the milk to various forms of sterilization or heating, in order that the contained bacteria may, for the most part, be destroyed, and that it may remain fit for feeding the infant for fully twenty-four hours. In places where the milk can be obtained direct from the dairy, and where we are certain that the collection of the milk has been carried out with care and with the especial object in view of feeding infants, we may do away with the heating process, especially in the winter time. In the summer, however, some form of sterilization is necessary.

Under the term sterilization the author includes both Pasteurization and sterilization.

Pasteurization.—Pasteurization is to-day the process most in vogue in this country for the preservation of infant food, and also to destroy, for the most part, any deleterious bacteria contained in the milk. It was first perfected by Pasteur, and therefore bears his name. The milk is subjected, in a suitable apparatus, to a temperature of 65° C. (149° F.) for a variable length of time, generally half an hour, and then rapidly cooled to 20° C. (68° F.) The most practical apparatus for this purpose was devised by Dr. Freeman, and is sold in the shops as the Freeman Pasteurizer. If properly carried out with this apparatus, the method destroys all pathogenic germs which may be present in the milk, and also a large percentage of the other bacteria of the milk, including most of the Bacterium lactis aërogenes, but does not destroy any sporulated bacteria, such as the Bacillus mesentericus vulgatus.

FIG. 19.

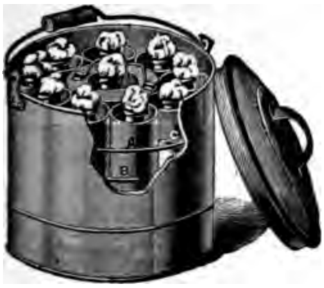
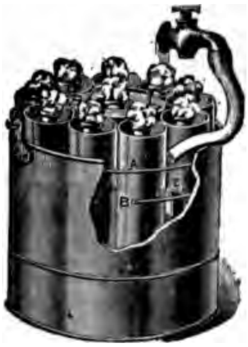


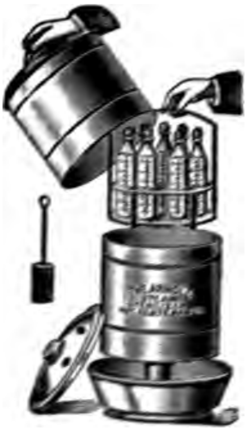
FIG. 20.



Freeman Pasteurizer.

Sterilization.—Sterilization is the process of heating milk to 212° F., or 100° C. This may be done by means of the Arnold Steam Sterilizer (Fig. 21), or by simply placing the milk in properly corked bottles in boiling water. As a rule, the milk is heated for twenty minutes, when it is considered sterilized. The milk should then be rapidly cooled, as in the process of Pasteurization, for by this process the fat of the milk will not separate. Sterilization is best performed by the above process, but the ordinary sterilizers will not render the milk absolutely sterile. It will not destroy any sporulated bacteria, but will destroy the Bacterium

FIG. 21.



Arnold steam sterilizer.

lactis aërogenes and all pathogenic germs. Milk which contains sporulated bacteria, such as the potato bacillus (Bacillus mesentericus vulgatus), may after a short time undergo a change due to the proliferation and action of the sporulated bacteria, which have not been destroyed by sterilization under ordinary atmospheric pressure. This consists in a splitting up of the casein and a so-called peptonization of the milk. This change begins after a few days, and when complete renders the milk alkaline in reaction and sweetish in taste. Milk, unless it has been sterilized under two atmospheres of pressure and at a temperature above that obtainable in the household sterilizer, is never completely sterile. Milk which has undergone the above peptonization is unfit for infant-feeding.

Disadvantages of Sterilization as Compared with Pasteurization.—In describing sterilization and Pasteurization of milk, it has been intimated that sterilization has its disadvantages, and these are, in short, that the lactalbumin of the milk is coagulated to a slight degree; the casein is changed, so that it is not as absorbable; the fats are liquefied, so that in sterilized mixtures they may be seen on the surface in the form of an oily layer; and the lime salts are converted into unabsorbable compounds, so that infants taking sterilized milk lose these salts for the economy. They do not get the necessary bone pabulum. This would account in part, if true, for the prevalence of scurvy in infants who take sterilized milk as an exclusive food for too long a period of time (Cronheim and Müller).

Though sterilization was at first a great step in advance, inasmuch as the process presented to the nursing infant the possibility of obtaining its food in a wholesome condition hours after its preparation, even in the hottest weather, there developed certain disadvantages in connection with its prolonged use. It has been noted, partly owing to the increased use of sterilized milk and partly to the fact that bottle-feeding has become much more general to-day than formerly, that infants who take sterilized milk to a certain extent do not thrive as well as infants who obtain either a mixed diet or a food not so thoroughly cooked. The result has been a decided increase in the number of scurvy cases, undoubtedly due to the changes in infant food. Aside from the danger of scurvy, a certain proportion of infants who do not develop scurvy and who are fed exclusively on sterilized milk remain stationary in weight, although the stools of such infants may be perfectly normal in appearance.

Again, infants who are taking sterilized milk develop in a certain proportion of cases inordinate constipation, and this in itself is a very troublesome feature in the feeding of these cases. In looking for another method of preserving the infant food, at least here in America, Pasteurization was next taken up. It was found, however, that the heating of the lactalbumin even to a temperature of 70° C. had its disadvantages, in that a certain amount of lactalbumin was coagulated. Still, the disadvantages of Pasteurization were less, as compared to those of sterilization, and it was at once apparent that if Pasteurization could be applied as a method of preservation of infant food, it would be a step in advance. The author at first advocated the heating of milk for infant-feeding at a lower temperature, a temperature subsequently taken up by Monti, of Vienna, of 180° F. At this temperature milk will keep twenty-four hours even in warm weather, with ordinary care, without turning sour. Even this temperature was found excessive, and Dr. Freeman advocated a still lower one for Pasteurization, and devised an instrument for carrying out this process, which to-day is in general use.

Coincident with the agitation against sterilization, and even Pasteurization of milk, the dairy methods have been so improved to-day that the time of Pasteurization can be reduced, and in midwinter, in large cities, the milk can be obtained in such purity as to be given raw to the infant. The whole question, therefore, of the preservation of milk has resolved itself into obtaining a milk as free from impurities and as recently from the dairy as possible. Thus, if we are certain of the cleanliness of our milk and the care with which it is handled, Pasteurization can be followed out as a method of preservation of the infant's food, even in the summer time; but such Pasteurized milk, no matter how clean the original milk when received from the dairy, must be kept carefully on ice in order to prevent its turning sour. Among the poor in large cities, however, Pasteurization is not safe in midsummer, and where large numbers of infants are fed from laboratories careful sterilization offers the best safeguard against infantile summer diarrhœa. In the fall and winter, pasteurization, in large cities is quite sufficient to preserve the infant food; and, as has been stated, in winter we may even, if we are sure of the source of our milk and its recency from the dairy, give raw milk to infants. Sterilization and Pasteurization, therefore, are simply methods of preservation of infant food, and have nothing intrinsic in themselves as regards the problems connected with infant-feeding.

Experimental Study of the Assimilation of Sterilized, Pasteurized and Raw Milk.

	Nitrogen taken in milk. Grammes.	Nitrogen remaining in feces. Per cent.
First infant—		
Pasteurized milk	10.9209	4.6
Sterilized milk	13.7449	4.9
Raw milk	5.3914	3.4
Second infant—		
Boiled milk	32.643	4.5
Sterilized milk	30.969	4.3

The table given above shows the comparative digestibility of raw, Pasteurized, and sterilized milk (Koplik), as indicated by the percentage of nitrogen remaining in the fæces of the infant. These experiments were performed by feeding the same infant with raw and heated milk. The results showed that, although the differences are slight, they are in favor of milk subjected to little or no heat. Doane and Price have confirmed these results by experiments on the calf.

What Shall the Practitioner do in Regard to Sterilization and Pasteurization?—If the patient has access to a milk which is only twelve hours from the dairy we may simply Pasteurize this milk both summer and winter, and in the summer-time it should be carefully kept on ice. During the winter we may give such a milk raw if obtained from a mixed herd of cattle. Raw milk from a limited herd is dangerous, inasmuch as the dilution is not great enough to eliminate impurities from sick cows, should there be such, in a small herd. The practitioner should therefore advocate a mixed milk from a large herd as the best safeguard against infection of the infant. The dairy should be kept scrupulously clean, as should also the animals, and the milk kept in clean utensils, in order that the above ideas may prove beneficial to infants. If the infant's milk (modified) is to be carried any distance during the summer, sterilization is a safeguard for a short period of time.

Raw Milk in Infant-Feeding.

With the improved methods of dairy hygiene and care exercised in most cities in the collection of milk intended for infant-feeding, the milk contains less bacteria and reaches the infant much earlier to-day than formerly. The result of this, at least in New York, where it is possible to obtain milk within twelve to twenty-four hours of the milking-time, has been that the milk is of a very low acidity and bacterial content. The question arises whether we may not give such milk, modified properly, in a raw state to the infant. For even Pasteurization, it must be admitted, tends to change the ingredients of the milk to such an extent as to compromise their nutritive value.

The author in practice Pasteurizes the infant's milk in the winter-time, and in many cases gives the milk in the raw state. In the summer, however, in large cities, where the icing of milk may have been imperfect, it is safest to sterilize the milk during the heated term. This is only for a period, at most, of three months. An infant taking sterilized milk under proper conditions during the heated term is not injured by such a food, and is protected from an attack of gastro-enteritis, for it is not possible, even though great care be exercised, to prevent an occasional bottle of milk from increasing in acidity. The result of such a change might be an attack of diarrhœa which would endanger life. In the fall, winter, and early spring the practitioner, if he is certain the milk is of good quality and has been collected in a careful and cleanly manner, need not do more than Pasteurize the milk. If he is absolutely certain of the source and freshness of the milk he may even give it raw. There are certain infants who have an idiosyncrasy against the taking of raw milk. The acidity cannot be rectified by lime-water, and the result is that such infants will have loose movements or even diarrhœa. These cases are exceptional, of course, but they must be borne in mind. In exceptional cases the author has seen even Pasteurized milk disagree in the same manner with the infant.

Moreover, we know now that the administration of heated milk, especially sterilized milk, over too long a period will cause bone disturbances, and it is certainly unwise to give, at least at the present day, sterilized milk to infants in the cooler seasons of the year. Even with the administration of Pasteurized milk for any length of time, it is well at about the fourth to the sixth month of infancy to give several times daily a small quantity of diluted orange-juice. In this way the ill effects of heated milk are counteracted, and the infant is supplied with those salts and acids which are lacking in the Pasteurized and sterilized fluid.

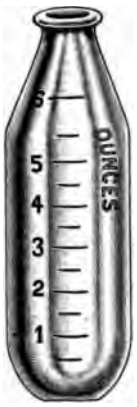
Frozen Milk.

The process of freezing is deleterious to cows' milk, inasmuch as it breaks up the original emulsion, and milk when thawed does not present the normal appearance under the microscope. The individual fat-globules are seen to be angular, and instead of presenting a spherical refracting body, the globule presents concentric rings, showing that in some way the cold has acted on the fat. Such milk, if given to an infant, will at times disagree and cause greenish diarrhœal movements, sometimes vomiting. Moreover, in midwinter it is very common for children who have previously been quite regular in their bowel evacuations, with movements of normal consistence and appearance, to become constipated as a result of the ingestion of milk which has been frozen and then thawed. It seems that the fat of the milk undergoes some change which interferes with its hitherto cathartic action on the bowels. As a result, these infants will have hard, constipated movements; or the movements may be partly constipated or partly of normal consistence. In such cases the physician will have no other resource but to advise patience until the milk can be delivered in an unfrozen condition.

Nursing Bottle.

The best form of bottle is the so-called Freeman bottle, which has very little neck, a wide mouth, not much shoulder to the neck, so that it may be easily cleansed. For newborn infants there is now constructed a very small bottle of the same model with a capacity of three ounces, the idea being that when milk is given in a small bottle, the heat is retained during nursing much better than when a small quantity of milk is contained in a large bottle. In the latter case the milk is chilled before the termination of the feeding. When filled the bottles are corked with non-absorbent cotton. They are corked loosely, so that the steam may escape. If the cotton is jammed tightly into the bottle, the cork will blow out in the heating. After nursing, the bottles are filled with a solution of washing soda and allowed to stand a few hours, and then washed externally and internally and drained dry. Any residue of milk remaining after nursing should not be utilized for another nursing.

FIG. 22.



Nursing bottle of the Freeman model.

The cleansing of the bottle is carried out with a so-called bottle brush. Nipples should be boiled once daily for ten minutes, and washed with hot water after each nursing. It is well to have several nipples carefully sterilized in the early morning and kept in a clean jar, rather than in a solution of boric acid. If the nipples are kept in boric acid the latter is apt to become contaminated, as also the nipples.

Before feeding, the bottle of milk is warmed to a temperature of about 100° to 105° F. (40.5° C.), so that the milk may not chill the stomach of the infant and thereby suspend the digestive process. Dr. Sobel has constructed a bottle-warmer, by means of which the milk may be heated to exactly the same temperature at every nursing. This is sold under the inventor's name in the shops.

IV. FOOD PREPARATIONS.

Peptonized Milk.—With the perfection of our methods of the modification of cows' milk, either in the laboratory or at home, the use of peptonizing agents as an aid to digestion of the casein of the milk has become more and more limited. On the other hand, it cannot be denied that the addition of peptonizing substances in safe quantities to the milk intended for the infant has a great advantage in certain cases of difficult casein digestion. As a rule, the infant will not take kindly to completely peptonized milk. It has a bitter taste, which cannot be overcome by the addition of sugar or any other agent to the milk. We are thus compelled, at least in the author's experience, to introduce the peptonizing agent into the milk in such a manner as not to change the taste of the food. The best method, therefore, of peptonizing the milk for infant-feeding is the so-called cold method. This is done as follows: The milk is modified, either at home or in the laboratory, in the ordinary way. Just before giving to the infant, if the amount is from four to six ounces at each feeding, one-fifth of a peptonizing tube is added to the mixture, which is then well shaken and placed in lukewarm water for two and a half minutes, aud then given to the infant. Such a milk will not have a perceptibly bitter taste.

Another method of peptonizing milk for infant-feeding is to employ the so-called peptogenic milk powder for this purpose sold in the shops. A bottle of modified milk containing four or eight ounces of the mixture is fortified with about an eighth of a measure of peptogenic milk powder just before feeding, heated for seven minutes in lukewarm water, and then given to the infant. Infants may be kept on this food for months, and then when the digestion and powers of assimilation have improved, the peptonization may be gradually omitted. The author has seen no ill effects from this method of giving peptonized foods. He feels, however, that at various intervals during the feeding of such infants, attempts should be made to omit the peptonizing ingredients from the mixture, in order to see whether the infant cannot thrive without them.

The indications for the use of peptonizing infant food will be given under the heading of Difficult Digestion.

Condensed Milk.—Condensed milk is very frequently employed to feed infants through the whole of the nursing period, and while it cannot be denied that some good results are thus obtained, condensed milk, pure and simple, for the majority of infants is not available. Many infants will cease to increase in weight under its continued use; others will develop rachitis and scurvy.

Condensed milk is sold in the shops in hermetically sealed cans, with or without the addition of sugar. The sugar is used to preserve the milk, and is generally cane-sugar. Condensed milk is poor in fats, although with the dilutions customary in infant-feeding, the proteids are not only low, but are in a more absorbable state than in most infant foods. Condensed milk also contains a very large proportion of sugar, both milk- and cane-sugar, and this, as has been pointed out under the heading of Nutrition, is one of the most easily absorbable foods for the infant.

An infant successfully fed on condensed milk will show a large deposit of fat. It may have a very good color, but a critical eye will invariably discover evidences of faulty metabolism, such as rachitis. Condensed milk is sometimes of great value in cases of gastro-enteritis, in which the digestion of ordinary modifications of cows' milk seem to be unsuccessful. It should only be used, however, in these cases to tide over a critical period. Condensed milk may be used fortified with cream, and under such conditions the cream is well assimilated. In traveling, also, if good milk is not available, infants who have been fed on carefully prepared mixtures may tide over a period of a few days on dilutions of condensed milk.

The following composition of condensed milk is given by König:

	Water.	Proteid.	Fat.	Milk-sugar.	Ash.
Condensed milk without cane-sugar	61.46	11.17	11.42	13.96	1.99
With the addition of cane-sugar 38.86 per cent.	26.44	10.47	10.07	14.16	2.00

In order to prepare condensed milk for infant-feeding, the milk is diluted ten to twelve times for infants below three months of age, and five to six times for older infants. In the cases of gastro-enteritis above mentioned, when the assimilation of cows' milk is difficult in the period following subsidence of symptoms, dilutions of condensed milk, with the cautious addition of raw cream or top milk, are borne better than modifications of cows' milk. This method of feeding should be resorted to only after a demonstration of the failure of milk modifications, and should only be preliminary to feeding with fresh cows' milk.

Barley-water.

Barley-water is one of the most useful adjuvants either to modified milk mixtures or as an exclusive food for a short time in cases of gastro-enteritis. The proper preparation of barley-water has been the subject of much study. The simplest method of preparing barley-water is that which utilizes the so-called Robinson's Patent Barley. A heaping teaspoonful of Robinson's Patent Barley is suspended in a pint of cold water until the lumps have disappeared. The mixture is then placed in a small saucepan over a gas-stove fire, and stirred constantly for fifteen to twenty minutes while boiling. The more the barley-water is boiled, the more thoroughly the barley is dissolved and dextrinized. After boiling, the loss in bulk is made up to the original quantity by the addition of water. The use of the so-called dextrinized barley instead of Robinson's Patent Barley offers in certain cases advantages to which reference will be made later on. Dextrinized barley is sold in the shops as such. It is made up of barley-pearls ground and heated for a long period of time according to the formula of J. Lewis Smith. The composition of Robinson's Patent Barley is given by König as follows:

Water	10.10
Proteids	5.13
Fats	0.97
N.-free extractives (carbohydrates)	81.87
Ash	1.93

It will be seen by a study of its composition that carbohydrates enter into it very largely. Fats and proteids are present in very small quantities. It is therefore unavailable as an exclusive food.

Oatmeal Gruel.—Oatmeal is utilized in the same manner as barley to dilute milk. It is made up in the form of a gruel. Two teaspoonfuls or more of oatmeal are boiled in a pint of water for twenty minutes and then strained. This decoction, made up in the same manner as the barley, is utilized to dilute the milk when barley has a constipating tendency.

The composition of oatmeal, according to Munk, is as follows:

Water	10.1
Proteids	14.7
Fat	5.9
Carbohydrates	64.7
Raw fibre	2.4
Ash	2.2

Arrowroot Gruel.—Arrowroot gruel has been used from time immemorial to dilute milk, especially in cases of summer diarrhœa. Dr. Merei is mentioned by Routh as having first suggested the use of this cereal for diluting milk. A teaspoonful or two of the arrowroot is added to a pint of water and boiled in the same manner as starch and oatmeal, strained, and the decoction used as a diluent with milk.

The composition of arrowroot, according to König, is as follows:

Water	16.50
Proteids	0.88
Fat	0.10
Carbohydrates	81.16
Raw fibre	0.05
Ash	0.19

Beef-juice.—The principal beef-juices are Valentine's, the preparation called Puro, Bovinine, Brand's, Wyeth's, Armour's, and Burgoyne's preparations of beef-juices. Beef-juices contain little protein and much extractive matter, so that the nutritive value is very low. There are some of these beef-juices, such as Bovinine, which are manufactured from blood rather than beef-fibre. In such a case the extractives are few and the proteids low; they are more in use than the other preparations. In order to take enough of these beef-juices to equal a teaspoonful of scraped meat in nutritive value, more must be taken than could be borne by the average stomach in illness (Hutchison). They are not, therefore, available as exclusive articles of diet for any length of time, and young children especially, whose palates are capricious, will rebel against most of these preparations, though they may prefer those which contain less salt than others. They are useful, therefore, only as articles of diet twice or three times in the twenty-four hours, and furnish ingredients in the shape of water and salts and very little protein to the body.

Composition of Beef-juices.

	Valentine's.[1]	Puro.[2]	Bovinine.[3]	Brand.[4]	Wyeth.[5]	Armour.[6]	Burgoyne.[7]
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Water	51.21	36.60	81.09	59.15	44.87	74.10	49.51
Proteids	9.65	30.33	13.98	15.45	38.01	8.30	13.00
Extractives	11.16	19.16	3.40	16.55		9.54	8.10
Mineral matter . .	10.84	9.79	1.02	8.85	17.12	7.51	14.20

Peptone Preparations.—By peptone preparations are meant such preparations as Somatose, Carnrick's Peptonoids, Fairchild's Panopeptone, and others. By referring to the table the reader will see that there are quite a number of preparations on the market. Of the peptonized foods in a ready form, the most concentrated by far is Somatose, which contains 80 per cent. of albumoses; whereas other preparations contain, as will be seen by reference to the table, very little proteid matter, and are, therefore, of very slight nutritive value. Somatose, however, though containing as it does the greatest

Showing the Composition of Peptone Preparations.

Preparation.	Water.	Soluble proteids (chiefly albumoses).	Extractives and other non-proteid organic matter.	Mineral matter.
	Per cent.	Per cent.	Per cent.	Per cent.
Somatose	9.20	80.00	. .	6.70
Carnrick's peptonoids . .	5.40	24.00	65.40 (mainly sugar)	5.20
Koch's peptone	40.16	34.78	15.93	6.89
Liebig's peptone[8]	31.90	33.40	24.60	9.90
Brand's beef-peptone . .	84.60	7.00	. . .	1.40
Denaeyer's peptone . . .	78.45	12.15	4.32	2.54
Darby's fluid meat[9] . . .	25.71	30.60	30.18	13.50
Armour's wine of peptone[10]	83.00	3.00	12.90	1.10
Fairchild's panopeptone[11]	81.00	6.00	13.00 (largely sugar)	1.00
Peptonized milk[12]	87.50	1.76	10.04 (=sugar, fat, and unaltered proteid)	0.70
Liquid peptonoids[13] (Arlington Co.)		5.25	12.63	0.95

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[1] Analysis by Dr. Candy.

[2] Fresenius (Leyden's Handbuch der Ernährungstherapie).

[3] Food and Sanitation, Dec. 23, 1893 (Analysis by Chittenden).

[4] Analysis by Dr. Candy (unpublished).

[5] The Lancet Analysis (quoted by the makers).

[6] Analysis by Dr. Attfield (supplied by the makers).

[7] Analysis by Dr. Candy.

[8] Leyden's Handbuch der Ernährungstherapie.

[9] *Ibid.* See also von Noorden, Therapeutische Monatshefte, June, 1892.

[10] Horton Smith's Journal of Physiology, vol. xii., p. 42, 1891, and Leyden's Handbuch.

[11] Maker's analysis.

[12] Horton Smith (*loc. cit.*).

[13] Maker's analysis also contains 14.94 per cent. of alcohol by weight.

amount of proteid matter, cannot be taken in large quantities for any length of time without causing diarrhœa, and in this respect it is unavailable as an exclusive form of food. In feeding infants and children I find it is of the greatest value in those cases in which it is necessary to give the stomach absolute rest and to feed per rectum. For such cases the Somatose is prepared as follows: A teaspoonful of Somatose is dissolved in eight ounces of cold water. Two ounces of this solution is given carefully per rectum, care being observed to pass the catheter above the second sphincter, in order that the food may not be rejected. This may be repeated every few hours. Thus given, a rectal enema is absorbed for the most part, and in some cases it may be mingled with milk part for part, the nutritive properties being thus increased.

Kumyss.—Kumyss has the following composition (König):

Water	90.44
Alcohol	1.91
Lactic acid	0.91
Milk sugar	1.77
Proteid	2.44
Fat	1.46
Ash	0.42

Originally kumyss was made from mares' or camels' milk by the addition of a ferment indigenous to Tartary, called kefir. To-day kumyss is manufactured from cows' milk by the addition of ordinary yeast-fungus, and contains, as will be seen by reference to the table, a certain amount of alcohol and lactic acid. I have never succeeded, even for a short period of time, in feeding infants on kumyss with any amount of satisfaction. It is only available in illness of older children with capricious palates. It use, therefore, is exceedingly limited. The same may be said of Matzoon.

Beef-extracts.—Beef-extracts are open to the same objections as beef-juices, in that they contain for the most part extractives and are not intended for prolonged periods of use. There are preparations, such as Bovril's, which contain meat-fibre, but which must be given in such concentrated form to obtain the necessary nutriment as to cause diarrhœa. Beef-extracts, on account of the warmth and contained salts, are supposed, when administered, to stimulate the appetite. A teaspoonful of Bovril's is equal to 8 grammes of lean meat, and therefore must be given in very large quantities, as stated above, in order to obtain any amount of nutrition.

Beef-broth.—Beef-broth has a composition of proteids 0.4, fat 0.6, salts 1.2, and extractives 1.2. With the extractives beef-broth contains creatin, xanthin, and hypoxanthin.

One pound of meat is cut up, placed in one pint of water, and allowed to stand four or five hours. It is then cooked over a slow fire for one hour. After cooling, the fat is skimmed off. This makes a very agreeable beef-broth.

Table Showing the Composition of Beef-extracts.[1]

	Liebig's extract.[2]	Bovril.[3]	Bovril for invalids.[4]	Armour's extract.[5]	Brand's essence.[6]	Vejos.[7]
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Water	18.3	44.40	21.82	15.55	87.17	25.02
Proteids	9.4	16.94	21.42	8.73	5.40	19.35
Gelatin		. .	. .	2.16	5.03	
Extractives . . .	30.0	20.32	39.60	43.23	1.01	21.02
Mineral matter .	23.6	18.32	17.16	25.91	1.39	14.07
Ether extract, etc.	18.6	. .	. .	4.12	. .	17.09 (Carbohydrate).

In addition to the above, beef-broth contains phosphate of calcium, earthy phosphates, sodium chloride, oxide of iron; the nutrition obtained from it depends mostly on the salts, especially of calcium combined with those of the phosphorus.

Acorn Cocoa.—Acorn cocoa is a preparation made in Germany, and may be obtained on sale in the shops. The author has found it of especial use in cases of diarrhœa and intestinal disease in which it is advisable to suspend the use of milk. It may be given for some days. Some children, however, object to its taste, and for this reason it is not applicable in every case. It contains fat, nitrogenous matter, and tannic acid. A teaspoonful of the cocoa is dissolved in eight ounces of water, and the preparation is given warm in much the same manner as milk.

Stohlwerck's acorn cocoa has the following composition:

Water (Fresenius, König)	5.28
Proteids	14.06
Fat	14.42
Sugar	25.15
Tannates	1.96
Extractives	23.39

V. ARTIFICIAL INFANT FOODS.

Infant foods have been not only the subject of much investigation, but the cause of very bitter feeling, both on the part of the profession and the manufacturer. Scientifically the physician is correct when he maintains that children cannot be brought up, as a rule, on the exclusive use of any infant food. On the other hand, it cannot be denied that infant foods in combination with cows' milk for short periods of time are very useful agents in tiding over many of the dyspeptic disturbances of infancy and childhood. It would be, therefore, incorrect either to discard infant foods or for the physician to regard them with suspicion. They are certainly useful, and it will repay the physician to try to understand the indications for their use, although it must be confessed that such indications are not always evident. An infant food, therefore, may be regarded as a carefully prepared article of diet, to be used with discretion in a limited number of cases of difficult feeding in infancy and early childhood. I do not hesitate to say that some of the most difficult cases coming under my observation have thriven on the judicious combination of an infants' food with cows' milk. We must understand, however, in giving the infant foods the exact indications in the case. They must nct be given empirically.

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[1] Hutchison, The Lancet, 1902.

[2] Analysis by Tankard.

[3] Analysis by Stützer (quoted by Voit, Münchener Medicinische Wochenschrift, No. 9, 1897).

[4] Analysis supplied by the company.

[5] Food and Sanitation, Dec. 16, 1893.

[6] Analysis by Dr. Candy (unpublished).

[7] The Lancet, April 16, 1898, p. 1060.

N. B.—"Vejos" is a purely vegetable product, but is included in this table for convenience.

The infant foods present to the practitioner either dried milk, a cereal in combination with it or alone, with or without the addition of a malt preparation of some kind. It is quite evident, therefore, that there are several serious objections to them as exclusive articles of diet for a great length of time. The principal objection is that they are dried or heated food substances. In a majority of cases this is a dangerous article to use for a prolonged period in infancy and childhood without combining it with some fresh article of diet, such as cows' milk.

Again, many of the infant foods contain nothing but a dry, carefully prepared cereal. It is evident that this alone cannot be given as an exclusive article of diet to an infant. It may be administered for a short time, as will be pointed out in the article on Infant-feeding; but it cannot be given for any prolonged period without giving rise to those very symptoms which we all fear in the feeding of infants, referable to the bones and the circulatory system; in other words, evidence of disturbed nutrition, such as rachitis and scurvy. Infant foods, therefore, if judiciously employed, are useful in the hands of a careful physician where other methods fail, but only as adjuncts to well-recognized forms of food, such as cows' milk.

We may divide infant foods roughly into three groups: The first group, such as Allenbury's, Horlick's, Carnrick's, and Nestlé's Food, contain cows' milk desiccated, combined with some cereal and sugar. These foods are intended as an exclusive diet for infants, and against these the scientist objects principally. They are foods which cannot be applied but in a very small percentage of cases as an exclusive food, and which if given over a prolonged period are open to the objections stated above.

The second group of infant foods are possibly the most useful, and are those which contain some form of malted carbohydrate.

The carbohydrates are in soluble form and the food may be regarded as a desiccated malt extract. Some of these preparations also contain diastase, and by combining the food with cows' milk or by the addition of some carbohydrate to the milk we can obtain a combination which is not only digestible for the infant, but may be of great nutritive value for a short period of time. In this group belong Mellin's Food, Lœflund's Malt Soup, the latter being nothing more nor less than the Liebig Malt Extract combined with potassium carbonate.

The third group of infant foods are those which are constructed of a pure cereal, and in this group are Ridge's Food, Imperial Granum, Robinson's Patent Barley, and others. This last group may simply be considered as very carefully prepared cereals. They are very useful in that we may apply them in those cases of intestinal disorder in which it is desirable for a short period of time to exclude milk completely.

If we study all these foods, including condensed milk previously mentioned, we shall see that they show a deficiency of fat and an excess of carbohydrates. On this ground alone their prolonged use is objectionable. The proteids present are either in the form of dried, heated proteids of cows' milk, one of the most indigestible forms of proteid substances that can be given to the infant, or in the nature of vegetable substances which are foreign to the infant dietary. Condensed milk also contains such an excess of sugar as to cause acid dyspepsia; although preparations of condensed milk are made up, as has been stated, without sugar. In the treatment of enteritis, both of the acute and subacute type, it is essential in very young infants to give temporarily some form of food which does not contain milk in any form. Although an ordinary cereal may be used in these cases, a more agreeable form is one of the infant foods, and especially Imperial Granum. This, made up to the consistence of ordinary barley-water, may be administered in cases of ileocolitis for quite a length of time, and will not be rejected by the infant or young child.

At the period of weaning—the ninth month—cereals may be added to the milk, in the form of an infant food, such as Ridge's Food, Imperial Granum, or barley. In such cases not only is the barley or infant food well borne, but the cereals aid digestion in breaking up the casein, which at this period is given in the milk in four times the quantity present in the human milk. Infants who suffer from colicky pains in casein digestion will be remarkably free from them if some form of cereal is added to the milk, and in some cases a malted food, such as Mellin's, added to the milk will aid digestion and set aside troublesome symptoms. It must not be forgotten also that in the malted foods, when added to the milk, we are giving a form of sugar, malt-sugar, one of the most digestible carbohydrates. This alone, in combination with other carbohydrates in the infant food, not only aids digestion, but also the increase in weight.

The objection raised to the combination of malted foods, starchy cereals, and milk, that the infant is not capable of digesting starch, does not obtain fully in practice. We find, as will be shown in case of the dextrinized gruels, that large quantities of carbohydrate and flour may be given to infants, and their digestion will not only be normal, but they will thrive and increase in weight very rapidly; whereas. under an ordinary milk diet they have remained atrophic.

Composition of Infant Foods.[1]

Food.	Water.	Proteid.	Fat.	Carbo-hydrate.	Mineral matter.	General description and remarks.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	
Dried human milk . .	. . .	12.20	26.40	52.40	2.10	**The standard of composition to which artificial substances should conform.**
GROUP I.						
Allenbury No. 1 . . . (For children before the age of three months.)	5.70	9.70	14.00	66.85	3.75	Desiccated cows' milk from which the excess of casein has been removed, and a certain proportion of soluble vegetable albumin, milk, sugar and cream added. No starch present.
Allenbury No. 2 . . . (For children from the age of three to six months.)	3.90	9.20	12.30	72.10	3.50	Resembles the above, but contains some malted flour in addition. No starch present.
Horlick's malted milk	3.70	13.80	3.00	76.80	2.70	A mixture of desiccated milk (50 per cent.), wheat flour (26¼ per cent.), barley malt (23 per cent.), and bicarbonate of soda (¾ per cent.). Contains no unaltered starch when mixed.
Carnrick's soluble food	5.50	13.60	2.50	76.20	2.20	A mixture of desiccated milk (37½ per cent.), malted wheat flour (37½ per cent.), and milk-sugar (25 per cent.). When prepared according to directions the casein is partially digested, but a considerable amount of unchanged starch is left.
Nestlé's milk food . .	5.50	11.00	4.80	77.40	1.30	A mixture of desiccated Swiss milk, baked wheat flour, and cane-sugar (30 per cent.). More than a third of the total amount of carbohydrate is in the form of starch.
Manhu infant food . .	8.86	8.70	5.60	75.90	1.00	A mixture of desiccated milk and malted cereals. When prepared according to directions contains a good deal of unaltered starch.
GROUP II.—*Class A.*						
Mellin's food	6.30	7.90	trace	82.00	3.80	A completely malted food. All the carbohydrates in a soluble form. May be regarded as a desiccated malt extract.
Class B.						
Savory & Moore's food	4.50	10.30	1.40	83.20	0.60	Composed of wheat flour with the addition of malt.
Benger's food	8.30	10.20	1.20	79.50	0.80	A mixture of wheat flour and pancreatic extract.
Allenbury malted food	6.50	9.20	1.00	82.80	0.50	A mixture of wheat flour and malt. When prepared according to directions it still contains some unaltered starch.

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[1] Robert Hutchinson, *Lancet*, 1902. (Modified.)

Composition of Infant Foods (Continued).

Food.	Water.	Proteid.	Fat.	Carbohydrate.	Mineral matter.	General description and remarks.
GROUP II.—*Class B* (*Continued*).	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	
Diastased farina . . .	8.30	7.60	1.30	81.70	1.10	**A malted farinaceous food. When prepared according to the directions, practically all the starch is converted into soluble forms.**
Coomb's malted food	7.90	12.10	2.80	76.80	0.40	**A malted farinaceous food.**
Nutroa food	6.80	15.90	10.30	66.00	1.00	**A mixture of cereals with the addition of a certain proportion of peanut flour, from which the somewhat bitter taste of the food and its high proportion of fat are derived.**
GROUP III.						
Ridge's food	7.90	9.20	1.00	81.20	0.70	**A baked flour, containing only 3 per cent. of soluble carbohydrates, the remainder being starch.**
Neave's food	6.50	10.50	1.00	80.40	1.60	**Resembles the above.**
Frame food diet . . .	5.00	13.40	1.20	79.40	1.00	**A thoroughly baked flour to which have been added cane-sugar and some extract of bran.**
Opmus food	10.90	9.10	1.00	78.60	0.40	**A granulated wheat food.**
"Falona"	7.00	8.40	3.50	79.90	1.20	**A mixture of cereals (oats, barley, and wheat), with a ground fat-containing bean.**
Robinson's groats . .	10.40	11.30	1.60	75.00	1.70	**Ground oats from which the husk has been removed.**
Robinson's pat. barley	10.10	5.10	0.90	82.00	1.90	**Ground pearl barley, poor in every element except starch and mineral matter.**
Chapman's whole flour	8.40	9.40	2.00	79.30	0.90	**A finely ground whole-wheat flour.**
Scott's oat flour . . .	5.80	9.77	5.00	78.20	1.30	**A fine oat flour.**
ADDENDA.						
Imperial granum . .	9.23	14.00	1.04	75.34	0.39	**(Classified under Group III.)**
Eskay's food	8.58	5.82	1.16	89.02	1.30	**(Classified under Group I.)**

VI. MATERNAL NURSING.

The ideal food for the infant is the milk of the *mother's* breast. Under our social conditions, the mother who can nurse her child from birth to the period of weaning is an exception to the rule, not because most mothers do not wish to nurse their infants. On the contrary, the author has found them very anxious to perform this function, but the average mother to-day has not the physical development that fits her to nurse the child. The result is that she cannot furnish sufficient milk, or that the milk is not of the quality requisite for successful nursing. Some mothers will have a sufficiency of so-called milk. The infants, however, do not gain in weight, are puny, have attacks of colic, and the symptoms indicate that the food is at fault. Examination shows that in such women true milk secretion is rarely established; the milk remains in the colostrum stage.

Some physicians think that if the infant cannot have the benefit of the maternal breast a wet-nurse is the alternative. If with the wet-nurse we had only to consider the fitness of the food, this would be true. If the maternal breast is not at our disposal, the next best and the safest thing for the race is a substitute for the breast, for many reasons, some of which we will try briefly to indicate.

In the first place, it is not moral nor conducive to the future good of the race to ask a mother (the wet-nurse) to put aside her own child and to deprive it of the breast for the sake of a strange child.

Second. No matter how healthy a wet-nurse may be at the time of examination, we have no assurance that such a wet-nurse will remain healthy, or that some diathesis not apparent at the time of examination may not be transmitted to the infant. We thus take a healthy infant, place it at a breast, and feed it with milk concerning the ultimate influence of which we are utterly in the dark. The author is inclined to believe that so far as human milk is concerned, certain tendencies may be conveyed from the nurse to the infant which will crop out later in life. By this he refers rather to scrofulous tendencies, lymphatic tendencies, tendencies connected with diseases of the blood-forming organs.

Third. The introduction of a stranger into the household is a cause of great disturbance to that household, and also one of concern to the physician. The idea that a child brought up at the breast is better fitted for the struggle of existence may be true; on the other hand, the difficulties, at least in this country, of obtaining fit wet-nurses for children are so great that it would be well, if the mother cannot nurse the infant, to place it on a substitute in the form of bottle-feeding, unless this is not feasible.

Of course, in all of this we do not include those exceptional infants which cannot be fed artificially. Such cases occur, and must be placed upon the breast.

Finally, if the mother can furnish two or three nursings daily, it is well *not* to take the child off the breast entirely, but to institute what is known as mixed feeding. In some cases this is a very satisfactory method of feeding the infant.

Contraindications to Maternal Nursing.

A mother may suffer from syphilis or skin eruptions or may have a deficiency of milk and under certain conditions may still be allowed to nurse her infant. A wet-nurse should be free from all constitutional and psychical taint to be fit to nurse an infant.

Syphilis can be communicated to the wet-nurse by the infant, or to the infant by the wet-nurse through luetic lesions of the nipple. A syphilitic infant, therefore, must not be allowed to nurse the breast of a woman who is free from syphilis; and we should be very careful not to place a child free from syphilis on the breast of a wet-nurse without previous careful examination as to the presence of syphilis in the nurse. A mother, on the other hand, who has syphilis can nurse her infant without danger of communicating syphilis to the infant if the mother has been exposed to and contracted the disease up to a period of two months before the delivery of the child. An infant congenitally syphilitic may nurse its mother without communicating the disease to the mother. These facts have been well established, and have been commented on in the chapter on Syphilis. Should the mother have contracted syphilis subsequent to the birth of her infant, and should she have been nursing the infant, it would be wise to take the infant away from the breast, for such a mother may communicate the syphilis to the infant in the same manner as a syphilitic wet-nurse.

Tuberculosis in the mother, if active, even in its milder manifestations, is a contraindication to her nursing her infant. Though the manner in which the toxins of the tubercle bacillus or the bacillus itself pass into the breast milk, if such be the case at all, is still a matter of study, we can well understand how the mother, weakened by the inroads of such a disease as tuberculosis, would be further seriously injured and weakened by nursing her child. The close contact of mother and nursling, furthermore, might favor the infection of the infant in other ways than by the milk alone. On the other hand, an old focus of tuberculosis, such as a healed pleurisy or coxitis long healed, in a vigorous mother would not contraindicate nursing should the secretion of milk be abundant and should the function not make inroads upon her health.

Active symptoms of Bright's disease, such as general anasarca and other signs of serious involvement of the kidney, would preclude a mother's nursing her infant, not only because such a function would weaken her, but because, metabolism being profoundly disturbed, the breast milk would be unfit for the maintenance of the nutrition of the infant.

Advanced disease of the heart would also unfit a woman for nursing her infant. On the other hand, a slight albuminuria not giving any objective or subjective symptoms should not interfere with the desire of the mother to nurse her offspring. Advanced and active disease of the liver would in the same manner as the above diseases contraindicate nursing.

Organic nervous disease with paralysis, severe neuroses, insanity, hysteria, epilepsy, neurasthenia of a marked type, when present in the mother, contraindicate the nursing of the infant. Aside from the disturbances said to be caused in the infant nursing the breast of a person the subject of hysterical or epileptic attacks, we would scarcely care to trust such a sufferer with the care of an infant. On the other hand, slight nervous tendencies in the mother should not contraindicate the nursing of the infant, for in such a case we would open the way for the deprivation of the breast to a large number of infants, and give an easy avenue of escape to some from the responsibilities of maternity. The severe forms of anæmia, leucæmia, malignant disease, such as carcinoma and sarcoma, the presence of a very marked goitre with active symptoms, may be mentioned as contraindications to the nursing of an infant.

The acute contagious diseases, the exanthemata, erysipelas; pneumonia, bronchopneumonia, pleurisy, acute rhematism, typhus and typhoid fever, diphtheria, are all contraindications to nursing the infant. A woman may be in the convalescent stage of typhoid and, if strong, may be allowed to nurse her child. I have seen mothers suffering from erysipelas nurse their infants without infecting them. This should not be the rule, however. In a case of diphtheria the danger to the infant of infection is much greater than would be counterbalanced by the benefits to be attained from continuance at the breast. The milk of a woman suffering from a severe pneumonia with a high febrile curve cannot be all that is desired for the infant, and the process of nursing with the accompanying physical and mental disturbance might react against the mother.

Selection of a Wet-nurse.

It is not necessary that the wet-nurse should have been recently delivered. A newborn baby may be given the breast of a nurse whose baby is from one to two months of age. In fact, her milk is preferable to that of a nurse who has just been confined. For, apart from the uncertainty as to whether the milk will agree with the baby, the milk after a few weeks attains a uniform composition, and is more likely to agree with the baby than milk from the breast of a woman recently confined. I prefer to place the newborn infant on a breast at least three weeks old.

The method of examining a wet-nurse as to her fitness begins with ascertaining the history of her own baby. It should sleep well in the intervals of nursing, be free from colic, and have normal movements. The baby should be completely undressed for examination. It should be at least tolerably well nourished. There should be no eruption on the skin, no copper-colored intertrigo, no snuffles, no pigmented spots, and no rhagades around the mouth or anus. The skin of the palms of the hands or the soles of the feet should not be fissured or hard or present suspicious pigmentation. The head should not have an idiotic, microcephalic conformity. The wet-nurse should be below the age of thirty. Old multiparæ do not, as a rule, furnish good milk. The shape of the breast is important. The pear-shaped, elongated, hanging breast furnishes more milk than the firm, round breast of virgin shape (Plate IV.). The nipple should be about one centimetre long and three-fourths of a centimetre in diameter. The baby can easily grasp such a nipple and draw it into the mouth. A flat nipple, or a nipple with fissures, or a nipple surrounded by eczema is not desirable in a nurse, and may even be dangerous to an infant. The nurse is next directed to undress, and her body is examined for traces of any eruption which may be specific. Pigmented macules should arouse suspicion, as also enlarged cervical or epitrochlear lymph-nodes. The lungs, especially the apices, are examined for bronchitis or tuberculosis. The nurse is rejected if there be the slightest evidence of apical involvement. The teeth should not be carious to such an extent as to preclude the possibility of their being kept clean. The presence of a fœtid ozæna is highly objectionable, apart from the offensive odor. Such cases may be latently tuberculous. The woman should be mentally sound. The wet-nurse is then examined as to the presence of venereal disease by inspection of the introitus vaginæ and the anus. The mucous membrane of the mouth should be examined for evidences of syphilis. Search is made for mucous patches and suspicious cicatrices. After having examined both child and mother in the manner detailed, we are in a position to recommend the nurse if the milk is satisfactory.

The physician should have at hand in his office means by which he can at once decide upon the desirability of a wet-nurse. He must not at the beginning be driven to the necessity of a milk analysis. He decides first as to the quantity and then as to the quality of the milk. As a rule, a wet-nurse comes to the physician insufficiently fed and in a frame of mind far from tranquil. If despite these conditions the milk possess the qualities desired, he may at once venture to place the baby at the breast. If the milk does not agree with the baby after a fair trial, future conduct will be guided by certain developments, both in the quantity and quality of the milk and the condition of the infant.

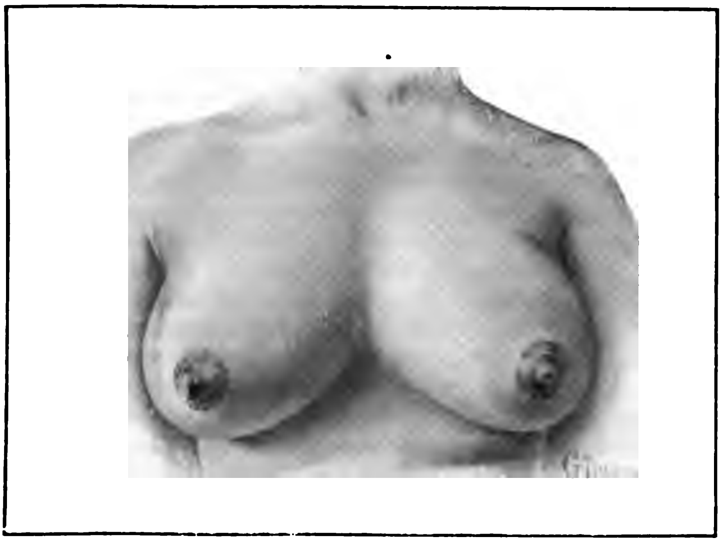
Quantity of the Milk.—The physician grasps the breast in the palm of his right hand and gently but firmly attempts to express the milk. The milk should with gentle pressure flow freely from the ducts. A drop is caught on the nail of the thumb. This time-honored nail-test is not to be despised. A drop of good milk will retain its bluish-white tint. This test will bring out the color of the milk, whether too watery, yellow, or white, to the experienced eye. The nurse is then directed to pump by gentle pressure a quantity of milk into a long, narrow beaker glass. If the breast has not been nursed within an hour, there should be no difficulty in obtaining at least an ounce of milk in this way. With this quantity we can at once decide on the efficiency of a nurse. The milk should have a bluish-white tinge. Any trace of yellow or green when a test-tube of the milk is held in the light, is abnormal. Milk may be very abundant but of a dirty white tinge; some specimens separate almost instantly upon withdrawal into a yellowish oily layer on top and a serous liquid below. Any such ab-

PLATE IV.

FIG. 1.

Form of the Breasts of a Wet-nurse with Abundant Milk of Good Quality. (After Schlichter.)

FIG 2.



Form of the Breasts of a Wet-nurse whose Milk is Deficient in Quantity and Quality. (After Schlichter.)

normalities in the milk should cause the rejection of an applicant. If the breasts, history, and physical examination are satisfactory, and the quantity and physical characteristics of a nurse's milk are good, we may recommend her without making a chemical examination of the milk. Such an examination is impracticable for the practitioner with the means at his disposal. Even if carried out, it may be unfair to the nurse. At the examining visit the proportion of proteids and fats may be below what it will adjust itself to in a day or two when the wet-nurse is rested and housed in her new home. More nutritious diet will greatly change the composition of the milk. There are, however, conditions which may require an examination of the milk at a subsequent period. In such a case the methods detailed elsewhere may be resorted to.

Once having determined to place the infant at the breast, the question arises, When should this function be begun? Immediately after birth the mother is tired and so is the infant. They have both gone through a critical period. It is well to let them rest for some hours. If the infant sleeps, and awakens only to be changed as to its diaper, we should not hasten to feed it. The author follows the rule that the infant be given a little water at intervals from the first six hours until the beginning of the next day after birth, and then the mother, having been thoroughly rested, the child is put at the breast, even though there are but a few drops of colostrum in the breast.

The first day after birth the infant should be fed at intervals of three hours. At this time there will be very little in the breast, but the stimulation of the breast by nursing will cause an increased secretion of milk, so that by the second day nursing may be inaugurated at regular intervals of two hours. After this the intervals of nursing are so apportioned that the newborn infant during the first week will obtain the breast from nine to ten times in the twenty-four hours; the second week, eight or nine times in the twenty-four hours; and in the fourth week, eight times in the twenty-four hours. After this the intervals of nursing will be much the same as they are in artificial feeding. We give the breast at intervals, generally of two and a half hours, so that the last nursing is at 11 P. M. After the first month the infant should sleep until five or six o'clock in the morning, when it obtains the first nursing. Then from the second to the sixth month seven nursings in the twenty-four hours are sufficient. The nursing should be so arranged that the mother and child may have complete rest of five hours between 12 P. M. and 5 A. M.

The number of times an infant should nurse at the breast is in the large majority of cases a matter of training and habit, especially with the breast-fed infant. Czerny, following, Ahlfeld advises placing the baby at the breast on the average of five times in the twenty-four hours. With care and patience this can be done. The practitioner, however, will meet a number of mothers who will nurse their offspring more frequently, and the above gives the limit of such nursings. In frequent nursing the infant receives less at each feeding than in the nursings at longer intervals.

The **care of the breast** really begins before the birth of the infant. About the seventh month of pregnancy colostrum appears in the breast. At this time it can be seen in some cases to exude from the nipple. Unless care is taken at this time we will have a fissuration of the breast nipple, due to the action on the epithelium of the skin of the drops of colostrum which are allowed to collect and to decompose on the nipple. The result is that at birth the mother may have sufficient milk in the breast, but be unable to nurse the child on account of the presence of these fissures. I advise, therefore, that at this time of pregnancy the nipples be kept scrupulously clean and washed twice a day with a dilute solution of alum water or some antiseptic wash. In this way the decomposition of colostrum on the nipple is avoided, and the nipple is strengthened by the slight massage of washing. If the nipple is not well developed, this is the time also to attempt its development. This is done by drawing out the nipple twice a day, either with the clean fingers or by means of suction. A small clay pipe may be used for this purpose, and the future mother may draw out the nipple by means of suction with this simple instrument. I am certain if this hygiene of the nipple is pursued that fissures of the nipple will be less frequent.

Fissured Nipples.—Ordinarily, if the nipple of the breast is kept dry and clean, it will not fissure and eczema will not occur. Fissures, however, sometimes occur even when great care has been taken to prevent them. Fissures or rhagades appear in about one-half of the nursing women. They are present either on the summit of the nipple or at its base. In the latter situation they are in the form of linear or circular ulcers. If fissures of the nipple are painful, the infant should not nurse the breast directly, but through a shield which protects the nipple, the best form being the Davidson shield. The fissure is painted once daily with a 10 per cent. solution of nitrate of silver. If there is a discharge of visible pus from the fissure, or if the breast nipple has a point of suppuration ever so small, the breast should not be nursed by the infant, for by so doing the mother may develop abscess of the breast or the infant may contract an infectious diarrhœa.

Physicians insist on placing infants at the breast immediately after delivery, for two reasons: first, because it is said that suction at the breast favors contraction of the uterus. Whether with this function there is contraction of the uterus has not been proved. Again, it is said that at this time suction will favor the flow of milk. Milk with colostrum does not appear to an appreciable amount in the breast, if not previously present, before twenty-four to seventy-two hours or even eight days after delivery. If, as has been pointed out, the breast is nursed too frequently, the traumatism caused by a vigorous infant will give rise to erosions of the nipple, and thus fissures. An excellent nursing breast may be ruined by overzealous efforts on the part of the physician. Fissures once present, if unyielding to the methods detailed above, must be allowed to heal by giving the breast perfect rest. Some women will nurse an infant at the breast, the nipples of which are the seat of fissuration, without pain, caking, or inconvenience. In other women caking will take place, with intense pain on nursing, and lymphangitis and abscess result. In all such cases of pain, lymphangitis, and caking nursing is best suspended, the infant being placed temporarily on the bottle. The breasts are supported, the fissures painted daily with silver, and if caking is present the breasts are emptied carefully with the pump and massage of the breast performed. If after the breasts become soft and the fissures are entirely healed there is still a little milk in the breast, the infant may be put again at such a breast, and if the organ is in a normal state the stimulation of suction will start a proper milk secretion. I have done this in a case in which the breasts had been at rest for three weeks after delivery, with excellent results. The milk returned in abundance, without unnecessary traumatism to the breast, the infant nursing only three times daily at first. We should never expose a mother to the danger of abscess of the breast by persistent attempts at nursing fissured nipples.

Caking of the Breast.—After the birth of the infant, the breast should be closely watched to prevent the so-called caking of the milk. If the infant is not strong and does not nurse well, there will be a residual amount of milk in the breast. After nursing, this milk should be pumped off with a breast-pump. The most satisfactory breast-pump is one with a glass bell and a rubber bulb. Pumping the breast at first, when the milk is forming, will prevent caking and rapidly regulate the secretion to the normal amount. On the other hand, if a fissure of the nipple is present, caking is more apt to occur, on account of the pain attendant on emptying the breast, either by nursing or by means of the breast-pump. We should be exceedingly cautious in these cases to examine the breast repeatedly in order that areas of caking may not escape us.

If caking occurs, the breast should be rubbed or massage performed three times daily. The hands of the nurse are carefully washed, and anointed with some sterilized oil. The breast is grasped in the palms of both hands, one above and the other beneath. The breast is then gently subjected to firm pressure with a vermicular motion. This massage is kept up for five or ten minutes.

Nursing the Infant.—The infant should nurse about twenty minutes and then fall asleep at the breast. The nipple is washed with a solution of boric acid before and after each nursing, and is covered in the intervals of nursing with a small piece of absorbent gauze folded several times. In this way the nipple does not come in contact with the clothing, and any exuding milk is caught on the gauze, which is replaced by a clean piece whenever necessary. The infant while nursing should lie in the arms of the mother or the nurse. The nurse grasps her breast just behind the base of the nipple with the index and ring fingers; the thumb should be used to exert pressure on the breast and thus regulate the flow of milk. In this way the infant is prevented from drawing the nipple too far into the mouth. The habit of moistening the breast with saliva or a few drops of milk is reprehensible. The infant's mouth will furnish all the moisture needed.

Signs of Efficient Breast-feeding.—An infant nursed at the breast is thriving if it has a good color, if its weight increases in regular ratio, if it sleeps between the nursings, and the stools are normal in color. It may be said in this place that, as to the stools, they will vary even in the most thriving infant, both in color and consistence, from time to time. An infant who is otherwise in good health and is not suffering from any disturbance of the gut will have from time to time slightly fluid, yellow movements; at other times the movements may contain a few whitish curds; and at other periods, even the most thriving breast-fed infants may show in the stools greenish discolored particles. If the infant shows no other signs of disturbance and is in good spirits, these changes in the color and consistence of the movements should not give us concern; they are dependent on the varying composition of the breast milk. If the milk contains on certain days more fat than usual, the movements may be softer and more frequent than customary. If the proteids are increased in quantity they may even show a greenish tinge. These conditions, however, must be infrequent and should not carry with them disturbances, such as colic, restlessness, or stationary weight. I have seen infants who were thriving, in that they had a very good color and their weight increased, but they suffered from inordinate colic, and examination of the breast milk showed, even at the second month of infancy, quite a number of colostrum corpuscles. After certain hygienic hints were carried out by the mother, these colostrum corpuscles disappeared from the milk, the colic abated, and the infant returned to a normal condition. Disturbances, therefore, of the gut are not always an indication for the cessation of *maternal* breast-nursing.

Signs of Inefficient Breast-feeding.—An infant is not thriving on the breast milk if its weight remains stationary for any length of time. For this reason infants should be weighed once a week at first, and after the second month at least twice a month. At the first indication of stationary weight an infant should be weighed every three days, in order to see whether there is any increase under new conditions. If the weight continues stationary the milk should be examined. It may be deficient in quantity to such an extent as to no longer satisfy the child. In that case the infant will be observed to nurse the breast for a long time, or it may nurse the breast a short time and then relinquish the nipple and cry; or it may cry in the intervals of nursing. All these are signs of inefficient feeding. In such cases the breast should be examined just before a regular nursing, in order to estimate the quantity of milk in the breast. The infant should be weighed, then given the breast, and weighed after nursing is completed. The breast is also examined after nursing. In this systematic way we can estimate the amount of milk taken by the infant at that particular nursing.

The movements of infants fed on an inefficient breast as to the quantity of milk are dry, constipated, and small. The author has seen the character of the stools improve upon increasing the quantity of food, either from the breast or by supplementing the breast with the bottle. In some cases the infant cries and has colic, the movements are passed with much flatus, and are uneven in consistence, lumpy here and there, with green discoloration. In such a case the quantity of the milk may be sufficient, but its quality is not up to the requisite standard. The nurse's milk should be examined not only chemically, but microscopically. A single chemical examination of the milk, as has been stated, gives no definite information. The milk, therefore, of the morning and evening nursings should be examined. It may again be emphasized that colic alone or combined with slight variations in color and consistence of the infant's stools is not a justification for the suspension of nursing. An infant may gain in weight, have good color, and still have inordinate colic. With patience and hygienic exercise on the part of the nurse colicky attacks will ultimately grow less frequent, and many infants who suffered colic at first will, as the second month approaches, cease to have colic as soon as the milk has definitely assumed a uniformly normal composition. Infants who thus have suffered colic at the second or third month after birth will cease to be inconvenienced and will thrive from this time forward.

If an infant at the breast fails to increase in weight, and at the same time suffers from inordinate colic, has green, curdy movements or a slight tendency to diarrhœa, it becomes a very important question as to whether it is not better to take such a child from the breast entirely, and to place it either on another breast or a substitute for the breast. An examination of the breast milk will aid us, as has been intimated elsewhere. If this breast milk reveals to any marked degree elements such as colostrum corpuscles and fails to show the characteristics of normal breast milk, we will still be more anxious to take such an infant from the breast. In fact, a continuation of an infant at such a breast is sometimes not devoid of danger. In one case the continued attacks of colic, accompanied by fluid movements, with green curds from birth, resulted ultimately in an attack of intussusception. This occurred in an infant five months of age. After the operation the infant was placed on the mother's breast again, and had a return of the former symptoms—constant colic, green curdy movements, alternating at times with slight diarrhœa. It was taken off the breast immediately, placed on an artificial substitute, and throve.

VII. MIXED FEEDING.

Mixed feeding is the administration of the breast, supplemented by the bottle containing some substitute for the milk lacking in the breast. Infants who are nursed on an inefficient breast as regards quantity of milk should be carefully weighed, and the quantity of milk in the breast estimated for the twenty-four hours. This may be done by weighing the infant before and after each nursing, or can be roughly estimated by simply observing the amount of milk that can be pumped off from both breasts combined two hours after a feeding. Having measured the milk, we can estimate within certain limits the amount of milk which such a breast would yield in twenty-four hours. If there is sufficient milk in the breast for two nursings, the mother should not be denied the pleasure of nursing her infant. We should not hastily reject such a breast as worthless, for two feedings of breast milk will be a great aid to the infant, both in the development of bone and the other tissues of the body. If two nursings exist in the breast, we would give the bottle six times in the twenty-four hours to an infant below the age of three months, and five times in the twenty-four hours to an older infant. In feeding on the bottle in combination with the breast, we should begin as we do in the newborn, with a low percentage of fats and proteids. Having accustomed the infant to the bottle, we should gradually work up to the normal percentage of fats and proteids, as will be shown in the chapter on the Feeding of Infants. The details as to the construction of the food are the same as those followed out with the infant fed upon the bottle exclusively. Care should be exercised in these cases to avoid overfeeding. Mothers are especially prone to overfeed infants, having an idea that a fat baby is a healthy one; but if it is explained to the mother that fat does not mean health, overfeeding may be avoided. This is especially true of mixed feeding; such infants are apt to be overfed and to be overweight, for a mother who has two nursings of the breast will be apt to consider this of very little moment and attempt to feed on the bottle, as if the infant had nothing from the breast at its disposal. The result is that such infants frequently suffer from overflow vomiting. In many cases this overflow vomiting does not seem to disturb the infant to any appreciable degree. It should be avoided, however, for we never can say when such vomiting may become a matter of serious moment.

VIII. ARTIFICIAL FEEDING OF INFANTS.

Artificial feeding of infants is the substitution for the breast milk of some one of the foods considered in the previous pages. Although attempts have been made to rear infants artificially on asses' or goats' milk, the experiment has failed, and cows' milk is universally utilized as a substitute for the mother's breast in artificial infant-feeding.

Before cows' milk can be given to the infant as a food it must be modified. By this is meant a dilution of the milk, as to its fat, proteid, and sugar constituents, so as to make it conform as much as possible, in percentages of its constituents at least, to human milk. There are two methods now well recognized of modifying cows' milk for infant-feeding. One of these methods is the so-called laboratory method of infant-feeding. The *laboratory method* of infant-feeding attempts to recombine the fat, proteids, and sugar of milk not only in proportions which conform to what is found in human milk, but to attempt to find out, by the frequent changing of these constituents, what is best adapted to each infant. Rotch and his school contend that what is good for or adapted to one infant may not be suitable for another. In his own words: "What is one infant's food may be another's poison." The Rotch method of infant-feeding has now had a very extensive and thorough trial. Its successes and failures will be talked of later on. Suffice it to say here that, in practice, if an infant does not thrive on a certain limited number of modifications of cows' milk, experience shows that we reach a point where our knowledge is no longer definite as to what changes may be required in the various formulæ in order to make the infant thrive. In other words, some infants will not thrive on cows' milk, no matter how many changes are made in the percentages of fat, proteids, and sugars. These are the difficult cases of infant-feeding which baffle the most skilful efforts at modifying cows' milk. The casein of cows' milk has been from the outset the point of attack. The old methods of infant-feeding considered simply the dilution of the whole milk two or three times, either with simple water or with some decoction of a cereal, either barley or arrowroot. In the first month the milk was diluted one in three; in the second month, one in two; in the third month, two in three, etc. These simple methods continued in use until Biedert, in Germany, and Meigs, in the United States, attempted to proportion the casein, fat, and sugar so as to make the mixture approach the composition of human milk. Biedert called his food a cream mixture. It was made in the same general way as Meigs' mixture. There was a low percentage of proteids, and a fat percentage corresponding to what is found in human milk. The proteids in Meigs' mixture ranged from 1.2 to 1.5 per cent. In Biedert's mixture the proteids existed to the extent of 1 per cent., fat 2 to 2.5 per cent., sugar 4 per cent. Meigs' mixture contains 3.5 per cent. of fat and 6 per cent. of sugar.

Biedert's Mixture.—Biedert took 50 ounces of milk, or 1.5 litres, and allowed it to stand one hour. The cream taken off the top of this milk contained 10 per cent. of fat. The amount of cream was 8 ounces. In other words, the top 8 ounces off 50 ounces of milk was a 10 per cent. top cream. With this he constructed the following formulæ.

Number of mixture.	Cream (10 per cent.).	Water.	Milk-sugar.	Milk.	Casein.	Fat.	Sugar.
	Litre.	Litre.	Grammes.	Litre.	Per cent.	Per cent.	Per cent.
I.	$\frac{1}{8}$	$\frac{3}{8}$	18	. .	(= 1.0	2.5	5.)
II.	$\frac{1}{8}$	$\frac{3}{8}$	18	$\frac{1}{16}$	(= 1.4	2.6	5.)
III.	$\frac{1}{8}$	$\frac{3}{8}$	18	$\frac{1}{8}$	(= 1.5	2.6	5.)
IV.	$\frac{1}{8}$	$\frac{3}{8}$	18	$\frac{1}{4}$	(= 1.8	2.8	5.)
V.	$\frac{1}{8}$	$\frac{3}{8}$	18	$\frac{3}{8}$	(= 2.1	2.3	5.)
VI.	. .	$\frac{1}{4}$	12	$\frac{1}{2}$	(= 2.3	2.4	5.)

If we compare these formulæ with Meigs' mixture, we find that Meigs contended that the infant needed through its whole nursing period practically one formula.

Meigs therefore had:

1. A 16-ounce top milk [7 to 8 per cent. of fat].
2. A solution of milk-sugar, 15 per cent.
3. A solution of lime-water.

He combined them as follows:

8 ounces { 3 ounces of top milk.
3 ounces of sugar solution.
2 ounces of lime-water.

This, according to our present methods, would give approximately a mixture of 3 per cent. of fat, 1.3 per cent. of proteids, 6 per cent. of sugar, which is also what Meigs strove for, with the exception that in some milks, as has been shown, more fat would be obtained than that given above, which is calculated from an average milk. With some milks Meigs obtained 4.7 per cent. of fat. To be more concise, Meigs designed the above method to obtain:

Water	87.6	Meigs' artificial food.
Fat	4.7	
Casein	1.1	
Sugar	6.2	
Salts	0.2	

In what follows we leave the reader to judge how much of Meigs' method still remains in the present modes of home modification of cows' milk.

It will be seen from the standpoint of to-day that both these men were the real pioneers of percentage feeding. It may be mentioned here that the method of Escherich is based on an attempt to calculate with rough dilutions of milk the amount of albumin necessary for the daily maintenance of nutrition. So far as the author knows, the Escherich method is very little in vogue in America.

The other two methods of modifying milk, which calculate the gross amount of calories necessary to maintain nutrition for infants, are the Huebner-Hoffman and the Soxhlet method. They have endeavored to construct a chemical mixture with the aid of cows' milk which is equal to the raw nutritive calories in mother's milk. In both these methods the milk is diluted with an equal amount of water. Huebner-Hoffman uses as a diluent a 6 per cent. solution of milk, whereas Soxhlet uses a 9 per cent. solution. The addition of sugar of milk is intended to take the place of fats, which are deficient in these mixtures. Sugar of milk, according to Soxhlet, has a caloric value equal to that of the fat deficit.

If it is desirable to feed a great number of infants in a public laboratory, I can say from actual experience that these mixtures are of the greatest utility, inasmuch as they can be easily prepared, and certainly the greater number of infants thrive on them. It is almost impossible in a laboratory intended for the use of the poor of a great city to give each child a percentage mixture. In other words, the feeding *en masse* is an entirely different problem from the feeding in private practice.

Infants from the first to the third month do not thrive as well on the Huebner-Hoffman and Soxhlet mixtures as they do on scientific modifications obtainable either in the laboratory or by the home method, which will be described. In other words, infants below the third month get in these mixtures an excess of proteids and deficiency of fat, and digestion is thereby impaired. The Meigs' mixture is most applicable to these cases.

The Rotch Method.—The method of Rotch has as its pivotal point the fact that all infants cannot be fed on the same mixture, and, taking the composition of human milk as a working basis, each infant should be considered as a separate problem in constructing a formula which within certain limits would be most suitable to its needs. Rotch therefore separates the milk from the cream by means of a separator or by gravity, and working with skimmed milk and cream containing 16 or 20 per cent. of fat and a dilution of milk-sugar, the constituents of the milk are rearranged. By this method an infant can be fed on a mixture of 1.5 per cent. of proteids, 3 per cent. of fat, and 6 per cent. of sugar; or 1.5 per cent. of proteids, 2.5 per cent.

of fat, and 6 per cent. of sugar, or any percentage of proteids, fat, and sugar that we may desire to give. Rotch also contends that an infant which may not thrive on 1.2 per cent. of proteids might do so on 1.5 per cent. The proportion of fat may be reduced or increased as needed in the individual case. In other words, the physician should consider his percentage formula in feeding the infant, just as he prescribes a certain strength of a drug.

To obtain these percentages a laboratory is needed, and to-day laboratories for supplying these mixtures to be used in the percentage feeding of infants are to be found in large cities. Though theoretically this method of reconstructing the milk would seem on the surface to be the most rational, it has certain inherent defects which Rotch and his pupils are trying to overcome. These defects are much the same as those of the older methods.

1. By simply rearranging the proteids, fat, and sugars we do not change the proportionate relationship which the casein or caseinogen bears to the lactalbumin and other proteids of the milk.

2. With the exception of a few limited facts and formulæ we have no data which, with our present knowledge, will enable us to know in every case when to increase or to diminish the proteids and also the fats.

3. The process of separating the cream from the milk by machinery destroys the original delicacy of the fat-emulsion in the milk. The infant does not assimilate these mixtures in every case as well as those which are constructed from milk which has not been manipulated to the extent that laboratory milk has.

In order to utilize the Rotch method by means of the laboratory, the physician has simply to prescribe the percentages that he requires on a slip made out for the purpose and furnished by these laboratories. It is needless to say that unless a physician is satisfied to follow a routine common to all his cases, instead of trying to understand the needs of each infant, he is certain to meet cases which even the most accurate modifications of the laboratory will not cause to thrive. In other words, the laboratory alone will not enable the physician to feed infants successfully. To do this he must know not only the percentages required at certain ages from constructed formulæ, but must study the digestion of each child, its movements, try to analyze from the movements whether the proteids are digested, or the fats are in excess or in diminished quantity. It may be said that in practice the most successful men find that they can get along on a very few fixed formulæ. An infant which will not thrive on these formulæ within certain limits will not thrive on any percentage modification of cows' milk, no matter how we may rearrange the percentages of its ingredients.

Principles Underlying the Rotch Method of Percentage Feeding.— As has been intimated, we must distinguish very carefully between infants who are quite normal and those suffering from disturbances of the gut in feeding them with cows' milk. The healthy infant needs but very few changes of formulæ throughout its infant life.

The first fact which we must establish from the birth of the infant is whether its organs are capable of digesting cows' milk at all. If such is the case, by a careful beginning and modifications of milk we can carry the infant along on very few formulæ, possibly three or four, through its period of infancy.

PROTEID.—The total amount of proteids in the cows'-milk mixtures must be very low for the newborn infant, certainly not to exceed 0.5 per cent. during the first week. After this the proteids are increased to 1 per cent., and kept at this point until the third month, when they are increased to about 1.5 per cent., and we may keep them here until the ninth month. For vigorous infants of heavy weight we may increase the proteids at the sixth month to 2 per cent. I have never found it necessary to go beyond that percentage.

FATS.—The fats in the first days after birth should be low—from 1.5 to 2 per cent. After the second week to the third month we may give from 2.5 to 3 or 3.5 per cent. of fat; rarely more than this. The reason for this is, that during this period the infant will not digest more fat. Infants who are getting a larger amount of fat than the percentage indicated will, as the nurse puts it, frequently "spit up" curds between the feedings. All the movements will be frequent, soft, and in some cases even of an oily consistence. In other words, infants who are taking a greater proportion of fat than that indicated will have a mild fat-diarrhœa, which may at any time become more severe and give rise to considerable concern. From the third month to the termination of infancy the fats may range from 3 to 3.5 or even 4 per cent.; never more than this.

SUGAR.—In modifying milk the sugars are placed in the mixture at a uniform percentage of 6 per cent. It is rare for us to be called upon to alter this percentage to any considerable extent. Too much sugar will cause in some cases fermentation in the gut, resulting in the production of gas. The children may thrive for a time on an excess of sugar; but in all these cases, sooner or later, a point is reached at which the sugar is no longer tolerated in large percentages. It is therefore unwise to give a larger percentage of sugar than that indicated.

SALTS.—The salts of the cows' milk are scarcely considered in percentage modifications. We know very little to-day about the fate of the salts in the cows' milk—how much of them are absorbed and exactly how much rejected by the infant gut. It has been intimated in another paragraph that the heating of the milk causes a complete loss to the economy of the salts present in cows' milk; but inasmuch as the heating of milk is coming more and more into discredit, and more pronounced efforts are being made to obtain a pure milk which can be administered with as little heating as possible, we have still to learn the fate of the salts in sterilized, Pasteurized, or raw milk, and the indications for adding equivalents of soluble salts to the milk for the feeding of infants.

A Schedule of Percentages Adapted to Infants of Various Ages.

Age.	Proteids.	Fat.	Sugar.
	Per cent.	Per cent.	Per cent.
Premature infants	0.33	1.00	5 to 6
One to seven days	0.50	1.50	5 to 6
Seven to fourteen days	0.80	2.50	5 to 6
Fourteen to thirty days	1.00	3.00	5 to 6
One to three months	1.25	3.75	5 to 6
Three to six months	1.50	3.00 to 4.00	5 to 6
Six to nine months	1.50 to 2.00	3.00 to 4.00	5 to 6
Nine to twelve months	3.05	4.00	5 to 6

Number of Nursings, with the Necessary Quantity of Milk to be Fed to the Infant.—The quantity of milk which should be given to the infant at each feeding from birth to the ninth month has been variously estimated. In the author's opinion and that of most practical men, the capacity of the stomach alone would be a crude and most unscientific standard, for this would not, in artificial feeding at least, follow nature's method with breast-feeding, for from birth the amount of milk furnished to the infant by the human breast daily does not always accord with the full capacity of the infant's stomach. It will be found that the quantity fed to the breast-fed infant is much below the stomach capacity if the infant is fed at frequent intervals, and, as has been shown in Ahlfeld's baby, equal to or even above it if nursed at long intervals. With artificial feeding, moreover, we know that there is a great waste in feeding infants upon cows' milk, and were an infant fed on exactly the same amounts of modified cows' milk as some of the breast-fed infants obtain from the breast, it would not increase regularly in weight and might even starve.

The age of the infant, also, is not a guide, for what would be a sufficient amount for one infant might not be sufficient for another, or might be even an excess. In all cases the capacity of digestion must be taken into account, and also the development of the child. Some vigorous infants will take more food than other infants of the same age that are not as well developed physically. More rational is the method of arriving at the amount to be given at each feeding which takes into consideration not only the capacity of the stomach, but the age and the amount of primary food elements necessary to maintain nutrition and to increase body-weight of the infant at various ages. If we calculate the amount of albumin or proteids or fat necessary per kilogramme of the body-weight to maintain nutrition, we shall have the more scientific method of determining the quantity of milk to be taken daily by the infant. This method has been advocated by Huebner and Rubner and also Escherich. The difficulty of calculating what is known as the calories necessary to the maintenance of nutrition and body-weight—and by calories is meant practically the amount of albumin or proteids, fat, salts, and water mentioned above—is, that the student or the physician cannot always have at his disposal a method by which these calculations can be made. In other words, they must rely on investigations made by others, and understand that the results as they are presented to us to-day in infant-feeding are not based on actual calculations of the amount of calories necessary to the infant. It has been found that the nutrition of artificially fed infants cannot be maintained by an amount of proteid of cows' milk equal to that taken in the breast milk. In other words, the proteid equivalent can be obtained, but other constituents, such as fat, would be at fault, as well as the daily quantity of food, were we to depend entirely upon the caloric method. The figures given to the student and physician to-day, therefore, are a combination of what has been found empirically to be needed, and what has been verified in the chemical laboratory to be absolutely necessary. Let the student therefore study the amount of breast milk consumed by the infant in the twenty-four hours, and compare these amounts with the amounts consumed by the bottle-fed infant in the same period of time.

Number of Nursings Daily with the Necessary Quantity of Each Feeding for the Artificially Fed Infant.—If we now attempt to apply the knowledge acquired in the study of the breast-fed infant to the artificially fed infant we meet with the following obstacles: Cows' milk taken in the same quantities, as has been said, is not as completely used up by the gut as breast milk. There is much more waste, as has been shown by Knöpfelmacher and Camerer. This waste is caused chiefly by the failure of the gut to assimilate completely the casein and the fat of the cows' milk. The stools, also, of bottle-fed infants are more numerous and of greater total bulk than those of breast-fed infants. Knöpfelmacher has shown that the waste of phosphorus of cows' milk is sixteen times as great in the gut as the waste of that element in the breast-fed infant. In view of the lack of definite knowledge on all these points, the quantities of modified cows' milk which should be given at each feeding to the infant are still, as has been intimated, only approximate. The amount of calories necessary for the maintenance of nutrition and a definite increase of the body-weight will be shown elsewhere, and the student may compare the tables given with the equivalent calories in the total amount of breast milk and cows' milk given to the breast-fed or artificially fed infant. He can therefore satisfy himself of this fact that the older authors, and even some of the most recent writers, underfeed their infants, if the food which they prescribe in their monographs and text-books is strictly adhered to in quantity and composition; and such is the fact, for many of these infants I found by observation not only to be underweight, but in some cases they fail in complete assimilation of their foods. Percentage feeding, therefore, is not as fixed and definite a science as the student would be apt to think were he to rely absolutely on the statements and formulæ of many writers. The student must also understand, however, that only a few of these formulæ and statements really epitomize the limit of our knowledge to-day, and future investigators must complete that knowledge.

Table Showing the Number of Feedings and Quantities of Modified Milk to be Given to Artificially Fed Infants.

Age.	Number of feedings daily.	Quantity at each feeding.		Total to be given in 24 hours.	
		C.c.	Oz.	C.c.	Oz.
First day	3	10		30	1
Second day	8	20		160	5½
Third day	8	30	1	240	8
Fourth day	8	40		320	10⅔
Seventh day	8–10	50		400	13½
Second week	8–10	60	2	480	16
Fourth week or first month .	8–9	60	2	480	16
Two months	7 or 8	90	3	630–720	21–24
Three months.	7	120	4	840	28
Four months	7	150	5	1050	35
Five months	6 or 7	180	6	1080–1260	36–42
Six months	6	210	7	1260	42
Seven and eight months . .	6	240	8	1440	48
Nine months	6	250	8½	1500	50

The increase in the amount of milk from the seventh to the ninth month is not so apparent, since at this period we, as a rule, begin to feed cereals in addition to the milk.

The above figures are not absolute, but only approximate. Some infants may require a half-ounce or more than the quantities indicated; others will be satisfied with less nursings. In all these items an observant student of the infant will, guided by the observations of the nurse of the infant, discover the indications in each case for himself.

Household Modification of Milk for Infant-feeding.

The accuracy of laboratory-feeding cannot be denied; its universal success, however, can well be questioned. For this reason a consistent attempt has been made to modify milk, as it comes from the dairy, in the home of the patient, without unnecessary manipulation. The accuracy obtained in home modification, it can be said, is as well adapted to the feeding of infants as the laboratory percentages. The author, whose experience is quite wide in the matter of feeding infants by home percentage modification, can say that the home modification of milk for infant-feeding, if carefully carried out, can boast of just as brilliant results as the laboratory method. The advantages of home modification of cows' milk for infant-feeding may be stated briefly as follows: The family and the physician can be independent of the modifier at the laboratory. The milk is manipulated as little as possible. If the infant does not thrive, we can say as definitely as by the laboratory method what is at fault. It has been shown that many of the laboratory modifications are no more accurate in themselves than those of the home method, and the percentage of error is just as great in the laboratory modifications, if carried out for a great number of infants, as the home modification. This has been shown quite conclusively by Townsend, of Boston.

The home modification of milk for infant-feeding depends on the fact that in large cities, and in places where milk is obtainable from the dairy within a reasonable time, the milk can be separated by gravity into top milk or cream and skim milk, and this separation takes place in certain definite proportions. Meigs, Biedert, and Chapin showed that it is possible to construct from top milk definite percentage mixtures, inasmuch as the top milk prepared in the manner to be described has an average constant percentage of fat, proteids, and sugar. Meigs was the first to work out a definite percentage mixture from top milk; Chapin has elaborated this idea.

Top Milk.—If milk fresh from the dairy is placed in a utensil, either a quart bottle or some similar vessel of like capacity, it separates within four hours after milking into a creamy, supernatant fluid and a skim milk. In the supernatant creamy fluid, or top milk, we find certain definite percentages of fat. In modifying milk in the home, the top layer as it separates from the milk is utilized as it is delivered in quart bottles. Chapin has found that if a number of milks delivered in the city homes are analyzed, the first 9 ounces from the top of the quart bottle of milk will contain all the way from 12 to 16 per cent. of fat, varying with the richness of the milk in fat.

Twelve Per Cent. Top Milk.—If the original milk contains 4 per cent. of fat, the first 9 ounces will be what is known as a 12 per cent. top cream. If the milk is a very rich milk containing butter fat to the extent of 5 per cent., the top 9 ounces will contain 16 per cent., approximately, of fat. The proteids are quite constant in the top milk and are equal to those found in the skimmed milk. In other words, in milk rich in butter fats the top milk contains fat in proportion to the proteids of 3 to 1. If the milk is poor and only contains 3 per cent. of butter fat, the first 9 ounces will contain generally 9 per cent. of fat, and this milk will contain 3 per cent. of proteids, so that the percentage of fat to proteids still remains 3 to 1. It may be said at the start that the student would do well not to consider the thin milk as existent, for most milk, either in the city or throughout the country, contains at least 4 per cent. of butter fat.

Seven Per Cent. Top Milk.—Another top milk to be considered is the so-called first 16 ounces taken from a quart of milk. If the milk is a rich milk and contains 5 per cent. of butter fat, the first 16 ounces will contain 9 per cent. of fat. If it is a rich milk and contains 4 per cent. of butter fat, the first 16 ounces will contain 7 or 8 per cent. of fat. The fat in both of these instances is present in a proportion of 2 to 1, as compared to the proteids. The student would do well to *assume* in making his modifications that he is dealing with a *rich* milk. In this way he will avoid giving mixtures which contain too much fat, which element gives the most trouble if present in too great quantity. If the student will therefore simply consider the top 9 and 16 ounces of rich milk, he will have sufficient material for feeding the infant up to the ninth month of infancy. The student should therefore try to perfect himself in the methods of utilizing top milk in which the fat is present, as compared to the proteids, in the proportion of 3 to 1, and a more dilute top milk in which the fat is present, as compared to the proteids, in the proportion of 2 to 1.

In feeding infants up to the third month it is convenient to use a top milk in which the fat is present, as compared to the proteids, in the proportion of 3 to 1. In other words, it is best to use the first 9 ounces of top milk, for by this method we can obtain, as will be shown by the tables, a smaller percentage of proteids and the requisite percentage of fat indicated in the earlier periods of infancy. From the third to the sixth month it is advisable to use a top milk in which the fat is present, as compared to the proteids, in the proportion of 2 to 1, for in this way we can obtain a larger percentage of proteids and more fat from one bottle of milk than we could if we use a smaller amount of richer top milk in which the fat is present, as compared to the proteids, in the proportion of 3 to 1, for in the latter case we shall be compelled to use 2 bottles of milk. This can more readily be understood by reading the subjoined tables indicating the percentages at the various ages.

Chapin, for the purpose of obtaining the top milk, has devised a small dipper. The use of the dipper is convenient but not necessarily essential. If the top milk is poured off carefully, equal accuracy is obtainable without the use of the dipper.

Top Milk Made at Home.

Clean Milk.—In cities milk is delivered in quart bottles, and in many places in the country this is also the case. But if the practitioner is living in a district where bottled milk is not sold or not obtainable, it is quite necessary that he should understand that there is no mystery about bottled milk. Any milk obtained shortly after milking and placed in a wide-necked bottle or utensil with a capacity of one quart will separate the top milk, or set, as it is called, in the manner previously described under the heading of Top Milk. This setting process takes place within four hours after the milk is placed in the utensil, so that, if the practitioner has not access to bottled milk, he can be just as accurate as the city physician if he will obtain an ordinary quart utensil, such as a pitcher, and place the milk in the same as soon after the milking as possible, setting it aside for four to six hours, and then proceeding according to directions given. Such milk will show the separation into the skim milk and creamy layer, as described elsewhere. There should be no visible dirt or dark specks in the bottom of the bottle, for such milk is unwholesome and should not be given to the infant. The milk on opening the bottles should have no peculiar odor, for such milk, no matter how carefully modified, will be rejected by the infant. If mixed with equal portions of 70 per cent. alcohol, milk when heated in a test-tube should not curdle. In other words, we should begin with a good, fresh, clean milk.

The Home Preparation or Modification of Milk for Infant-feeding.

In what follows it must not be forgotten that the formulæ and statements are directed toward the management of distinctly normal cases. If an infant does not thrive on these formulæ, the physician must be thrown on his experience and deftness in order so to modify the formulæ that digestion will be accomplished without difficulty. We will consider the percentage modification of cows' milk in the household, presupposing that there are no difficulties in the way of the infant's complete assimilation.

The Method of Calculating Percentages.

We know that in the first 9 ounces of top milk the ratio of fat to proteids is as 3 to 1, and in calculating any percentages, whether we fix on the proteids or on the fats as a method of calculation makes very little difference, provided we remember this proportion. For example: If we calculate on a formula containing 3 per cent. of fat, and we desire to construct this formula with the first 9 ounces of top milk, the proteids in that formula will be 1 per cent. If we wish to give 0.25 per cent. proteids from the first 9 ounces of top milk, the fat must necessarily exist in a percentage of 0.75. It is well, therefore, for the practitioner simply to fix in his mind what percentage of one or the other ingredient he desires to give to the infant, calculate upon that, and the fat or proteid will exist in that formula in the ratio indicated. The author, for convenience, fixes the amount of proteid which he wishes in his mixture, multiplies that by 3, to obtain the percentage of fat that would exist in that mixture, and proceeds in the following way: An infant at birth, for example, will receive 0.5 per cent. of proteids, its fats would be 1.5 per cent., if constructed from the first 9 ounces of top milk.

Let us suppose, for example, that a 12 per cent. top milk is to be used, and that the total amount to be given in twenty-four hours is 8 ounces. We wish to reduce the percentage to 1.5. The question involved is, "How much of the 12 per cent. top milk must be used to make a 1.5 per cent. 8-ounce mixture?" The following mathematical statement simplifies the process:

If of a 12 per cent. top milk you would use 8 ounces in twenty-four hours, to make a 1 per cent. top milk you would use $\frac{1}{12}$ of 8, equal ⅔ ounces. To make a 1.5 per cent. top milk you would use 1.5 times ⅔, equal 1 ounce.

One ounce, then, of a 12 per cent. top milk, diluted 7 times, will give an 8-ounce 1.5 per cent. mixture.

How to Work Out the Above Percentages of Fat, Proteids, and Sugar.—PROBLEM 1.—Let the physician take, for example, a premature infant. By referring to the schedules it is seen that such an infant should have 10 or 12 feedings in the twenty-four hours. The most assimilable mixture should have a strength of 0.33 per cent. proteids, 1 per cent. of fat, and 5 or 6 per cent. of sugar. Such an infant should have 12 feedings, each ½ ounce, making a total of 6 ounces for the twenty-four hours. If a 12 per cent. top milk is utilized, inasmuch as the fat-percentage of our mixture is 1 and that of our top milk is 12, the total quantity in the twenty-four hours being 6 ounces, we need $\frac{1}{12}$ of 6, equal ½ ounce of this 12 per cent. top milk, which must be diluted by 5½ ounces of water or barley-water, as the case requires, in order to obtain a mixture of 6 ounces containing 1 per cent. of fat.

In order to get the requisite percentage of sugar of milk which, when mingled with the diluent and the ½ ounce of top milk, will approximate 5 per cent., 2 teaspoonfuls of sugar of milk should be dissolved in the diluent before adding the top milk.

PROBLEM. 2.—The infant is one month old. Such an infant would assimilate best a mixture approximating 1 per cent. of proteids, 3 per cent. of fat, and 5 per cent. of sugar. It would need 10 feedings in the twenty-four hours, each containing 2½ ounces, making a total quantity of 25 ounces. If the 9-ounce top milk is used (12 per cent. of fat) we would proceed as follows: The percentage of fat desired being 3, and the total daily quantity being 25 ounces, we would have to take $\frac{3}{12}$ of 25, equal to $6\frac{1}{4}$ ounces of 12 per cent. top milk, with $18\frac{3}{4}$ ounces of the diluent, which should contain 6 per cent, of milk-sugar, or 7 teaspoonfuls.

Problem 3.—The infant is four months old, and it is desirable to construct its formula from the 16-ounce top milk (7 per cent. fat), ratio of fats to proteids 2 to 1. The percentages most adapted at this age would be 3 of fat, 1.5 of proteids, and 5 of sugar of milk. This infant should have 8 feedings in the twenty-four hours, each containing 5 ounces, a total of 40 ounces of food in the twenty-four hours. The percentage of fat being 3, that of the top milk 7, and the total amount of food being 40 ounces, there would be needed $\frac{3}{7}$ of 40, equal to 17 ounces of top milk, with 23 ounces of the diluent, to which is added 6 per cent. of milk-sugar, or 9 teaspoonfuls.

For the above formula it will be necessary to use 2 bottles of milk, taking 16 ounces off each, mixing them together, and of these 32 ounces to utilize 17.

Problem 4.—The infant is six months of age, and would need 7 feedings, of 7 ounces each, making a total of 49 ounces for the twenty-four hours. The formula most adapted in this case would be 3 per cent. of fat, 1.5 per cent. of proteids, and 5 per cent. of sugar of milk, utilizing the top 16 ounces of a bottle of milk, the percentage of fat in the formula being 3, that of the top milk 7, and the total amount of the food being 49 ounces, there would be needed $\frac{3}{7}$ of 49, equal to 21 ounces of top milk; 28 ounces of the diluent will be necessary, containing 5 per cent. of milk-sugar, or 9 teaspoonfuls.

It will be necessary in this case, also, to utilize two quart bottles of milk to obtain 21 ounces of 16-ounce or 7 per cent. top milk. That is, 32 ounces of this top milk are obtained, and of these 21 ounces only are utilized.

Problem 5.—The infant is nine months of age. In this case 6 feedings will be given in the twenty-four hours, each containing 8 ounces, making a total of 48 ounces. The formula most adapted to this age would be 4 per cent. of fat, 2 per cent. of proteids, and 5 per cent. of milk-sugar. The percentage of fat being 4 in the formula, that of the top milk 7, and the total quantity of food for the twenty-four hours being 48 ounces, the physician would need $\frac{4}{7}$ of 48, equal to 24 ounces of 7 per cent. or 16-ounce top milk, 24 ounces of the diluent, and enough sugar of milk to make a 5 per cent. solution.

Problem 6.—An infant six months of age, for therapeutical reasons, is to be put on a formula containing 1.5 per cent. of fat, 0.5 per cent. of proteids, and 5 per cent. of sugar. Here the percentage of fats to the proteids is as 3 to 1, therefore it will be convenient to use the top milk containing 10 to 12 per cent. of fat and 3.5 per cent. of proteids. It is desired to give the infant 7 feedings of 7 ounces each, making a total of 49 ounces. The percentage of fat being 1.5 in the formula, and that of the top milk being 12, the total quantity for the twenty-four hours being 49 ounces, the physician would need $\frac{1.5}{12.0}$ of 49, equal to $6\frac{1}{8}$ ounces of top milk, $42\frac{7}{8}$ ounces of the diluent. In order to get a 5 per cent. solution of the milk-sugar there would be needed in this case 5 per cent. of 42 ounces, equal to 18 teaspoonfuls of the milk-sugar.

It frequently happens with infants above three months of age taking a modification of the 16-ounce top milk that constipation will set in, and we wish to increase the fats in order that the movements may be less constipated. In order to do this we must obtain a top milk which is richer in fat than the top milk we are giving. To illustrate: The infant who is taking a third dilution of the 16-ounce top milk will be taking approximately 2.5 per cent. of fat, 1.2 to 1.5 per cent. of proteids. If we wish to increase the fats to 4 or 3.5 per cent. and retain the proteids we are administering to the infant, it will be impossible to do this with the 16-ounce top milk, for any dilution of this milk will vary the proteids. We are therefore compelled to resort to the utilization for such an infant of the 9-ounce top milk, which contains an average of 10 to 12 per cent. of fat. By diluting this one-third we would get about 3.5 to 4 per cent. of fat and still retain the same percentage of proteids as in our original mixture.

An infant four months of age, taking eight bottles, 5 ounces each, would need 40 ounces for its daily mixture. We would therefore be compelled to use, in order to obtain the 9-ounce top milk, 2 quarts of milk, from each of which 9 ounces would be taken, making 18 ounces of top milk. This, after being thoroughly mixed, would be utilized to the extent of 13 ounces for our mixture, giving 27 ounces of the diluent, whatever that may be, we would have a formula of 3.5 per cent. fat, 1.3 to 1.5 per cent. proteids.

Formulæ constructed with top 9-ounce milk, having an average composition of 12 per cent. fat, 3.5 per cent. proteids, 4 per cent. sugar. Possible combinations.

Fat.	Proteid.	Sugar.
1.00 per cent.	0.33 per cent.	5 per cent.
1.50 "	0.50 "	5 "
2.00 "	0.66 "	5 "
2.50 "	0.83 "	5 "
3.00 "	1.00 "	5 "
3.50 "	1.20 "	5 "
4.00 "	1.33 "	5 "
4.50 "	1.50 "	5 "

Formulæ constructed with top 16-ounce milk, having an average composition of 7 per cent. fat, 3.5 per cent. proteids, 4 per cent. sugar. Fats to proteids 2 to 1. Possible combinations.

Fat.	Proteid.	Sugar.
1.00 per cent.	**0.50** per cent.	5 per cent.
1.50 "	**0.75** "	5 "
2.00 "	**1.00** "	5 "
2.50 "	1.25 "	5 "
3.00 "	**1.50** "	5 "
3.50 "	1.75 "	5 "
4.00 "	**2.00** "	5 "

Whole milk having an average composition of 4 per cent. fat, 3.5 per cent. proteids, 4 per cent. sugar. Fats to proteids 8 to 7. Possible combinations.

Fat.	Proteid.	Sugar.
1.00 per cent.	**0.85** per cent.	5 per cent.
1.50 "	1.32 "	5 "
2.00 "	**1.60** "	5 "
2.50 "	2.15 "	5 "
3.00 "	**2.60** "	5 "
3.50 "	3.00 "	5 "
4.00 "	**3.50** "	5 "

It should be understood that the percentages of fats given in these tables can only be obtained with approximate accuracy, for there is no milk which will yield an absolute fixed percentage of fat in the top milk obtained by gravity, without variation, from day to day. The proteids, however, are more constant in percentage; but even here in modification we can only obtain approximate accuracy. Though these tables contain 8 modifications each, some of them differing but $\frac{1}{4}$ of 1 per cent. either in the fats or the proteids, such minutiæ are not really needed or even possible in practice. It will be found best to master 3 or 4 modifications of top milk, constructed either from the 9-ounce top milk or the 16-ounce top milk, and utilize these in general practice. For example: The infant who is taking 1 per cent. of fat and 1.33 per cent. of proteids may do just as well on 1.2 per cent. of fat and 1.50 per cent. of proteids. For all practical purposes, therefore, formulæ which contain 1.5, 2.5, and 3.5 per cent. of fat will be as available in practice as formulæ containing 1, 2, and 3 per cent. of fat.

Referring to the proteid percentages, it will be seen that certain of them are in heavy-faced type. Both in the laboratory and at home it is impossible to obtain an accuracy which will assure the physician that he is administering to his patient 0.66 and not 0.5 per cent. of proteids, or some intermediate figure; nor can he be certain that his mixture, even if prepared at the laboratory, contains 1.23 or 1.33 per cent. of proteids, rather than some slightly higher or lower figure. The reason for this is that the proteids of cows' milk, like, the fats, must vary from day to day, and thus no absolute fixed average percentage of proteids can be counted on. König, in an analysis of several hundreds of milks obtained from a number of *herds* of cows, shows that the proteid percentages in milk vary, not only at different seasons of the year, but at times of the day, and also with different kinds of fodder. It is therefore illogical to attempt the working out of minutiæ of percentages varying from 0.2 to 0.3, when the original milk has not a fixed average percentage. To obtain accuracy within the difference between 0.2 to 0.3 per cent. would necessitate a chemical analysis of the milk before each modification is made, a manifestly impracticable procedure, especially as regards the proteids in the milk. The author has gone into these matters to show that the elaborate tables given by some enthusiasts are, on careful analysis, impracticable. It is well, therefore, for the physician to feel assured that with the proteids, as with the fats, approximate formulæ with averages of 0.25, 0.5, 1, 1.5, 2, and 2.5 per cent. of proteids are as effective in practice as minute fractional percentages, if such were attainable. In practice, assimilation cannot be reduced to that delicate percentage accuracy insisted on by some authors.

Too High Fat-percentages and Their Remedy.

If Problem 4 is studied it will be seen that 2 bottles of milk must be utilized in order to obtain the requisite 21 ounces of top milk, and if this is so for the sixth month, more of this top milk will be required for the seventh and eighth months. Some infants will not thrive on such a large amount of fat. In the summer especially they will spit up, and have several loose movements daily. In the face of such difficulties I follow the plan of using only 1 bottle of milk; and, if after the fifth month (Problem 3) more than 16 ounces of top milk are required, I take these off the top of the bottle, adding the rest as diluent. Thus, at the sixth month, 21 ounces off the top of a quart of milk to 28 ounces of diluent. At the seventh month, 23 ounces off the top of a quart of milk to the required amount of diluent. At the eighth month, 25 ounces off the top of a quart of milk to the required amount of diluent. The amount of diluent is calculated as in the former tables.

By this method of simply increasing the amount of milk taken off the top of *one* quart of milk after the sixth month, we arrive at a point (the tenth or eleventh month) when the infant is taking a full quart of milk with diluent daily. This method, which is exceedingly simple, and which in summer particularly does away with the danger of excess of fats, has served me well.

If this method is pursued, the strict calculation of percentages of fats and proteids is necessarily abandoned.

PROBLEM 7.—Let us suppose that for certain reasons top milk cannot be obtained, or the milk obtainable is whole milk and the people are not sufficiently intelligent to construct top-milk mixtures. In the table of possible combinations with whole milk there is a most available formula:

Two per cent. of fat; 1.6 per cent. of proteids. Whole milk having a strength of 4 per cent. of fat.

7 feedings are needed.
$\underline{\ 7}$ ounces each.
49 ounces in the whole mess.

Percentage of fat needed, 2, divided by 4 per cent. in the whole milk will result as follows:

$$\frac{2}{4} \times 49 = \begin{cases} 24 \text{ ounces of milk,} \\ 25 \text{ ounces of diluent.} \end{cases}$$

PROBLEM 8.—Taking the same infant with the same 49-ounce mixture to construct the formula:

Three per cent. of fat; 2.6 per cent. of proteids. We would need:

$$\frac{3}{4} \times 49 = \begin{cases} 37 \text{ ounces of milk,} \\ 12 \text{ ounces of diluent.} \end{cases}$$

Diluent.—Very little has been said thus far as to diluents in modifying cows' milk. The principal function of diluents is to dilute or cut up the casein of the milk, or caseinogen, and at the same time dilute the fat to such a degree as to make both these ingredients more digestible in the infant stomach. As diluents used in modifying cows' milk, a solution of milk-sugar of definite strength, barley-gruel, or whey is used.

Solution of milk-sugar should be 5 or 6 per cent. strength. Milk-sugar chemically pure is sold in the shops as such, and it is dissolved in water which has been filtered and boiled or in distilled water.

As to barley-water, the preparation of which is detailed in full elsewhere, it should be remembered that the milk-sugar is dissolved in the barley-water while it is being boiled, as in this way there is no residue.

Reaction.—Lime-water is added to all milk mixtures in order not only to make them more alkaline, but to aid, as has been shown, in the digestion of the casein by delaying coagulation of casein in the stomach and favoring the passage of the milk or stomach contents into the intestine. The food should contain, according to Meigs and Rotch, from one-twentieth to one-twenty-fifth of its bulk of lime-water.

Lime-water is made by adding about an ounce of unslacked lime to half a gallon of boiled or distilled water, shaking well, and then allowing it to stand until the supernatant liquid is clear. It is then ready for use.

Lime-water is best added to the food just before giving to the infant. Thus, to an 8-ounce mixture are added 3 teaspoonfuls of lime-water. I generally advise the omission of the lime-water after the sixth month of infancy.

Summary Table of Infant-feeding.

Age.	Percentage of proteids.	Percentage of fat.	Percentage of sugar.	Daily feedings.	Amount of each feeding.	Total daily quantity.	Top milk to be used.	Amount of top milk.	Milk sugar.	Diluent.
First day . . .	0.30	1.50	6	3	10 c.c.	30 c.c. 1 oz.	9 oz. or 12 per cent.	ʒj or 3.9 c.c	ʒss or 1.5	ʒvii or 26.5 c.c.
Second day . .	0.50	1.50	6	8	20 c.c.	160 c.c. 5½ oz.	9 oz. or 12 per cent.	℥v or 20 c.c.	ʒij or 8	℥iv ʒv 140 c.c.
Third day . . .	0.50	1.50	6	8	30 c.c. or 1 oz.	240 c.c. 8 oz.	9 oz. or 12 per cent.	℥j or 30 c.c.	ʒiij or 12	℥vij 210 c.c.
Fourth day . .	0.50	1.50	6	8	40 c.c. or 1 oz.	320 c.c. 10 oz.	9 oz. or 12 per cent.	℥j ʒiij 40 c.c.	ʒiv or 16	℥ix ʒiij 280 c.c.
Fifth day . . .	0.50	1.50	6	8–10	40 c.c. or 1 oz.	320 c.c. 10 oz.	9 oz. or 12 per cent.	℥j ʒiij 40 c.c.	ʒiv or 16	℥ix ʒiij 280 c.c.
Sixth day . . .	0.50	1.50	6	8–10	40 c.c. or 1 oz.	320 c.c, 10 oz.	9 oz. or 12 per cent.	℥j ʒiij 40 c.c.	ʒiv or 16	℥ix ʒiij 280 c.c.
Seventh day . .	0.80	2.50	6	8–10	50 c.c. 1½ oz.	400 c.c. 13½ oz.	9 oz. or 12 per cent.	℥ij ʒv 80 c.c.	ʒv or 19	℥x ʒv 320 c.c.
Fourteenth day	0.80	2.50	6	8–10	60 c.c. or 2 oz.	480 c.c. 16 oz.	9 oz. or 12 per cent.	℥iij ʒiij 100 c.c.	ʒv or 22	℥xij ʒv 380 c.c.
Fourth week . .	1.00	3.00	6	8–9	60 c.c. or 2 oz.	480 c.c. 16 oz.	9 oz. or 12 per cent.	℥iv 120 c.c.	ʒv or 21	℥xij 360 c.c.
Two months . .	1.25	3.75	6	7–8	90 c.c. or 3 oz.	630 c.c. 21 oz.	9 oz. or 12 per cent.	℥vj ʒiv 196 c.c.	ʒvj or 25	℥xiv ʒiv 484 c.c.
Three months .	1.25	3.75	6	7	120 c.c. or 4 oz.	840 c.c. 28 oz.	16 oz. or 7 per cent.	℥xiv 420 c.c.	ʒvj or 25	℥xiv 420 c.c.
Four months .	1.50	3–4	6	7	150 c.c. or 5 oz.	1050 c.c. 35 oz.	16 oz. or 7 per cent.	℥xv 450 c.c.	℥j or 36	℥xx 600 c.c.
Five months .	1.50	3–4	6	7	180 c.c. or 6 oz.	1260 c.c. 42 oz.	16 oz. or 7 per cent.	℥xxiv 720 c.c.	℥j or 30	℥xviij 540 c.c.
Six months .	2.00	3–4	6	6	210 c.c. or 7 oz.	1260 c.c. 42 oz.	16 oz. or 7 per cent.	℥xxiv 720 c.c.	℥j or 30	℥xviij 540 c.c.
Seven and eight months	2.00	3–4	6	6	240 c.c. or 8 oz.	1440 c.c. 48 oz.	16 oz. or 7 per cent.	℥xxiv 820 c.c.	℥j ʒiv or 43	℥xxiv 720 c.c.
Nine months .	2.00	4.00	6	6	250 c.c. or 8½ oz.	1500 c.c. 51 oz.	16 oz. or 7 per cent.	℥xxix 870 c.c.	℥j ʒj or 37	℥xxj 630 c.c.

When is a Bottle-fed Infant Thriving?

It may be said that a bottle-fed infant is thriving if it increases regularly in weight, wakes up betimes to nurse the bottle, does not suffer from colic, and has movements of uniform consistence and color. It should not "spit up," as it is said, to an inordinate degree. There should be no rejection of food after the bottle has been given, thus showing that the quantity has been accurately gauged. The color of the infant should be good. The young infant should sleep most of the time, except when nursing or engaged in play. Older infants should have a happy, contented expression of the face.

We do not consider an infant with a very large deposit of fat as necessarily a healthy one. On the other hand, another of exactly the average weight may be much healthier than the infant who is overweight. Thus, the physician will have to draw conclusions from various data of color, weight, development, and well-being of the child as to whether it is thriving on the food mixture.

Physicians should not be afraid to leave well enough alone with the artificially fed infant, and, if the gain during some weeks is not up to the standard, should not be discouraged, in view of the fact that the succeeding week may show the average gain. Bottle-fed infants gain very irregularly; sometimes for a week may appear to have gained but very little, an ounce or two. The succeeding week may show a marked recuperation and gain in weight above the average.

The physician imbued with the principles of percentage feeding also should not be too hasty to change percentages, but should endeavor to content himself with a *minimum number* of changes. In this way the parents of the infant will be impressed with the fact that the artificially fed infant is not taking, even at the best, a perfect food, but only one which must make up the deficiencies caused by the lack of the mother's milk.

Among the disturbances from which apparently normal infants suffer, and by this we refer to infants who are thriving, are, first, constipation. The physician will see an infant on one mixture have two movements daily, perfect in color and consistence; whereas another infant on the same mixture will be inordinately constipated, and movements hard, having the form and consistence of scybalæ. He will thus learn to account for this constipation on various grounds. A certain percentage of newborn infants are apt to be constipated, and this constipation is due to an inherent inertia of the gut, and also a lack of secretion of the normal lubricating fluids of the gut. In these cases also we may find a tendency to constipation inherited from the mother.

Given an infant with constipation, there are various modes of rendering this symptom a matter of less care to the physician, as well as to those in charge of the infant. If the food is heated, it is well to omit this process or to reduce the heating to the minimum. We should endeavor not to give constipated infants sterilized food; but rather, in the winter and fall, Pasteurized or raw food. By food we mean milk mixtures. In some cases it is necessary to increase the amount of fat in the mixture, and in this place we should caution the physician to go very slowly in increasing the fats for the purpose of overcoming the constipation. If the fats are increased we should never, especially with the newborn or young infant, give more than 3 to 4 per cent. of fat. In many cases 4 per cent. of fat will be followed by other symptoms fully as annoying as the constipation which the increase of fat was intended to remedy. I refer to the so-called spitting or rejection of part of the food after nursing. This consists in the bringing up of a number of curds in the intervals between feedings. These curds, as a rule, have a sour odor and are accompanied by eructations of gas. In such cases it is best to reduce the amount of fat, for in very young infants an irritation of the stomach to any marked degree, as evidenced by the rejection of a part of the food in the spitting of curds, may result in serious vomiting, a symptom much more to be feared than the constipation. However, in the administration of top milk we very often find, especially with the newborn (and by newborn I refer to infants below three months of age), that a fourth dilution of top milk replaced by a third dilution will often remedy the constipation. Fat-diarrhœa caused by too great a percentage of fat will be taken up later.

Spitting.—Spitting, or rejection of part of the food after nursing to any extent, may become an annoying symptom, and the physician should try his best to remedy it, although the infant may apparently be thriving. A breast-fed infant may spit to quite a degree and not cause us any uneasiness; but it is otherwise with an artificially fed infant. Such a condition may lead to serious enteric disturbances, necessitating a suspension of the food entirely; or the spitting may be due in some cases to an excess of fat, and we should try with such infants, even though thriving, to reduce the fat gradually until we arrive at a point at which the spitting is less evident, at the same time retaining the percentage of proteids in the mixture.

Colic.—Bottle-fed infants who are apparently thriving and at times quite contented will have one or two attacks of colic in the twenty-four hours. In a breast-fed infant we may have a number of colicky attacks. The breast-fed child may thrive, the movements may not show much change from the normal, and the physician in these cases is not disturbed; on the other hand, in an artificially fed infant an excessive degree of colic is a cause of uneasiness, not only to the family, but to the physician, for it indicates that the digestion of the proteids in the mixture does not proceed along physiological lines. It has been stated that one or two attacks of colic daily are not inconsistent with perfect health in the infant, if the movements are of normal consistence and color. On the other hand, any excess of colic, combined with a disturbance of the consistence and color of the movements, the appearance of curd particles or white curds in the movements, or a yellow movement containing too much fluid, mixed with white curds, is a signal for a change in the percentages of the mixture. In most of these cases the proteids are at fault, and they must be reduced. We should not, however, reduce them to too low a figure. An infant one month of age can normally digest 0.8 to 1 per cent. of proteids. An infant three months of age may digest 1 to 1.5 per cent. of proteids; an infant six months of age may digest normally 1.5 to 2 per cent. of proteids. Other infants will find it difficult to digest, even at the sixth month, 1.5 per cent. of proteids, and will have less colic and the movements will be more consistent and normal in color on 1 per cent. of proteids. Less than 1 per cent. of proteids for an infant from three to six months of age will result in a diminished gain in the weight of that infant, although the infant may be thoroughly comfortable. On the other hand, some infants at the age of six to nine months may digest 2 per cent. of proteids without the least discomfort, so that working between these limits the physician will have to find out the amount of proteids that can be completely digested by the infant, always bearing in mind never to allow the proteids to reach too low a percentage, else not only diminished gain in weight will result, but also other disturbances of nutrition which we wish to avoid.

Fat-diarrhœa.—The physician, while increasing the proportion of fat in his mixture, the infant thriving at the same time, will find that the movements will at times become fluid, though yellow in color; and at other times will be more consistent and of the same color. With some infants the movements will become of an oily consistence. In such extreme cases there will also be uneasiness with the movements, and colicky attacks. Movements which are normal in color, contain no curds, whose consistence is of an oily character, indicate that the fats are in excess of the necessary quantity. Such infants may even gain in weight on this excessive amount of fat. The food should be suspended in these cases for a few hours, and the mixture administered with a diminished amount of fat. Such infants will do well on low fat-percentages; whereas other infants of the same age will take more fat and still give no evidences of fat-diarrhœa.

Greenish Movements.—Bottle-fed infants, apparently thriving, will have at times movements which contain green residue and white curds, and this will be followed by a movement which is perfectly normal in color and consistence. This may be repeated at intervals of a week, and I am accustomed to lay no stress on such an occurrence. On the other hand, if such green movements occur frequently and are accompanied by colicky pains, it indicates that the milk is not digested and that the proteids are at fault. Such infants can scarcely be included in the normal category ; they are simply mentioned here, and the subject will be taken up in another section.

Disturbances in the Bottle-fed Infant which are on the Boundary Line between the Normal and the Abnormal.

(1) **Vomiting.**—Some mothers will tell the physician that the child vomits a certain amount of its food once or twice daily and does not seem to be very much disturbed by it. If such an infant increases in weight, looks well, and has movements of normal consistence there is very little indication for our interference, except, perhaps, to reduce slightly the amount of food administered at each nursing. The cases, however, which puzzle the physician are those which vomit 2 or 3 times daily, and which do not increase in weight in a physiological ratio. Such infants increase slightly in weight at first, and after a time cease to increase. We have then to deal with an abnormal condition.

(2) **Too Low a Percentage of Proteids.—Stationary Weight.**—It has been mentioned that the physician should be cautious not to reduce the percentage of proteids beyond a certain limit. If he does, the child will not only fail to increase in weight, but the development of the child will be below the normal, and we may even incur the danger of scurvy, pronounced rachitis, and other evidences of disturbed nutrition.

(3) **Too low a percentage of fats** will also result in disturbed nutrition to the infant. Constipation is one of the results of too low a percentage of fat. By this we refer to a percentage of 1.5 of fat for an infant five months of age. If such a percentage of fat is continued for two or three months, the infant will cease to increase in weight and will develop those disturbances of nutrition already mentioned.

(4) **Assimilation of the Food Without Increase in Weight.**—It is not infrequent, especially in the newborn, to find infants who completely assimilate the mixture we administer to them. They sleep well, are not disturbed by colic, the movements may be constipated or of normal consistence and color, and still the infant fails to increase in weight. These are the baffling cases. An increase in the percentages for the newborn infant, or in the quantity of the mixture, can be made within certain limits. If we overstep the bounds, the mixture will disagree with the infant and cause symptoms which will necessitate a temporary suspension of the food.

When Shall the Food be Peptonized?

It has been mentioned elsewhere by the author that in peptonizing the food he makes use of only one method—the cold method—for the reason that most infants will not object to the taste of the food when this method is employed. The cases in which an attempt should be made to peptonize the food are as follows: The newborn infant is placed upon a percentage mixture. It suffers from constant colic, sleeps very little, has movements which are green, mixed with curds; on the whole the infant remains stationary in weight or the increase is very slight. In these cases most satisfactory results are sometimes obtained by peptonizing the food in the following way: Just before the food is administered a third or a quarter of a so-called peptonizing tube is added to the milk; it is well shaken and heated for two minutes. With this exposure to warmth there is very little development of the bitter taste in the milk. It is then given to the infant. It is surprising to see what an immediate change occurs in the general condition of the infant. The child will sleep, the pain and colicky attacks disappear, the movements become yellow in color and normal in consistence; the increase of weight will begin and continue along physiological lines. The physician must not expect, however, that this result will follow in every case, or that this method of preparing the food excludes every attempt to attain success by varying the percentages of proteids and fats in the milk. It is to be supposed that before any attempt is made at peptonizing the mixture the physician has made every effort to find the correct proportions for his particular patient, and having satisfied himself that the percentages are correct and that there is a difficulty in the digestion of the proteids, he may proceed to peptonize the milk, but not under any other conditions.

Whey Method of Modification of Cows' Milk.

This method is really very old. In Routh's "Infant-feeding" we have the whey method, similar to what is practised to-day, described by Mr. Lobb. This gentlemen, in a brochure on hygiene, read before the Harveian Society, gave the details of preparing a "compound resembling human milk," and this mode of modification of cows' milk as devised by Professor Falkland. Recently the method has been taken up by Vigier, and elaborated by Monti, of Vienna, in 1897. Rotch has advocated this method of diluting milk for feeding infants with difficult digestion.

Whey has a composition, according to König, of—

Proteid	0.8	per cent.
Fat	0.2	"
Sugar	4.7	"
Lactic acid	0.3	"
Salts	0.6	"

The proteid contained in whey includes the lactalbumin of the milk and lactoprotein. The salts are potassium sodium, lime, and magnesium, with iron in combination with chlorine, phosphorus, and sulphuric acid.

Whey is made by adding 1 part of rennet to 200 parts of milk at a temperature of 35° to 40° C. (95°–104° F.), or a tablespoonful of the rennet sold in the shops may be added, roughly speaking, to a quart of milk, allowed to stand, mixing thoroughly until the milk separates into a liquid and a curd portion. The curd is then broken up thoroughly and the whole is strained through cheesecloth. About 20 ounces of whey may be thus obtained from a quart of milk. The rennin of the rennet is still existent in the whey, and must be destroyed before the whey can be mixed either with milk or cream for the purpose of modification. In order to do this the whey must be heated to the temperature of 165° F., at least—that is, Pasteurized—for thirty minutes. Older authors advocated bringing the whey to a boil. Whey, as such, without the addition of cream or milk, is exceedingly useful in feeding infants who are suffering from enteric catarrh. It contains, as is seen, the liquid proteid substances of the milk, with salts and water. An infant can be kept on such a diet for several days without the danger of being starved. It has certain advantages over albumin-water, which will be described later. It is acid in reaction, and may be sweetened with sugar if the children object to taking it.

The principle of its introduction into infant-feeding, combined with certain percentages of cream, is founded on the fact that, when we modify cream or milk to make it conform to the formula as found in human milk, we are still dealing with a casein which is not present in proportion to the lactalbumin as it is in human milk. By thus separating the liquid proteids from the casein and recombining them this disparity of percentage is overcome.

The proportion, as has been stated before, of the casein or caseinogen to the remaining proteids of cows' milk—the lactalbumin and lactoglobulin—is five-sixths of casein to one-sixth of lactalbumin and lactoglobulin, as compared to human milk, which contains two-sixths of caseinogen and four-sixths of lactalbumin and lactoglobulin. In the whey we obtain all the absorbable proteids; and if we use cream, which is highly concentrated, for fat-proportions and skim milk to obtain the caseinogen, we can make a mixture which both relatively and actually contains the same proportions of caseinogen, lactalbumin, and lactoglobulin as human milk. It must be said at the start, however, that the preparation of milk modified by the whey method is carried out with the greatest difficulty at home; and even when constructed at the laboratory the method has not yet been perfected to such an extent as to be entirely devoid of objection. It very frequently happens that unless the whey is thoroughly and most carefully Pasteurized, the modified milk curdles when heated. It is very difficult thus to prepare the whey mixture, although, theoretically speaking, it seems to be the ideal method. It has not come into vogue for the reason that the physicians have not yet accustomed themselves to the theory of preparing these solutions. It is also found that the manipulation to which the milk is subjected is open to the same objections that ordinarily obtain with modified milk as prepared at the laboratory. Children, for some reason not yet explained, do not thrive as well on these carefully prepared mixtures as they do on mixtures prepared in the ordinary way. This does not at all invalidate the fact that the method may be so perfected that a certain class of cases may be fed by this method. White and Ladd have reduced casein in these mixtures so that, with concentrated cream, skim milk, and whey, they obtain mixtures in which caseinogen and casein, as stated, bear the same proportions relatively to the lactalbumin and lactoglobulin as it does in the human milk; that is, with a total proteid percentage of 1.25, two-thirds are whey proteids and one-third caseinogen.

The following table shows a few of the combinations of caseinogen and lactalbumin obtainable from the laboratory:

Fat.	Caseinogen.	Lactalbumin.	Sugar.
1.00 per cent.	0.25 per cent.	0.25 per cent.	4 to 7 per cent.
1.50 "	0.25 "	0.75 "	4 to 7 "
2.00 "	0.50 "	0.75 "	4 to 7 "
2.50 "	0.50 "	0.75 "	4 to 7 "
3.00 or 3.50 "	0.50 "	0.75 "	4 to 7 "

The method is of so little vogue that we have still to await data as to whether infants thrive on it.

IX. WHEN SHALL THE PHYSICIAN RESORT TO INFANT FOODS.

Under the heading of Infant Foods have been indicated the conditions under which these foods may be utilized. No conditions there laid down presuppose that any infant food may be used as an exclusive diet for the infant. Infant foods are only either a temporary makeshift—where milk for some reason must be excluded from the dietary—or they may be added to milk to aid its assimilation. In the first set of cases belong the infant foods which have been indicated under the heading devoted to this subject, such as Imperial Granum or the carefully prepared cereals. These foods are used in forms of dyspepsia or intestinal disease to tide over a critical period. To the latter class belong the infant foods of the malted varieties, such as Mellin's Food, which are added to the milk to aid its assimilation. In other words, we utilize the diastase or malted sugar to aid in the digestion of the proteids of the milk. Inasmuch as this aspect of the subject has been fully gone over in a proper section, we refer the reader to that section.

Barley-gruels and How to Utilize Them.

Some physicians object to the addition of barley-gruel in any strength to the milk intended for the normal infant, on the ground that the gut of the infant is not prepared for the assimilation of starchy food, and we find authorities who deprecate the use of barley-gruel for the newborn infant, on the ground that it is difficult of digestion. We find others who deprecate the use of barley-gruel under all conditions other than actual disease. The author's experience does not carry out the assertion that barley-gruel is not well borne by the newborn infant. On the contrary, some of the most successful cases of infant-feeding in his case-book are those of newborn infants whose percentage mixture contained as a basis a thin barley-gruel. These cases are especially those newborn infants with whom the digestion of the proteids is very difficult. He makes up the barley-gruel for these infants as follows: A heaping teaspoonful of Robinson's Patent Barley is always allowed to a pint of water. This is dissolved, then stirred over a gas-flame, brought to a boil, and kept at this temperature for fully fifteen to twenty minutes. While the barley-gruel is boiling, the amount of milk-sugar requisite for the infant's mixture is added. The gruel is then allowed to cool, and the top cream is added in the requisite percentage quantity. If prepared in this way, we will have greater success than with a barley-gruel only momentarily heated to the boiling-point. Milk mixtures prepared in this way have a consistence of thin gruel and are quite well borne, not only by the newborn infant, but throughout the nursing period. The use of so-called dextrinized barley in the making of the gruel, on the other hand, is not well borne by younger infants, inasmuch as there is a greater residue and the solution is not as complete as with the ordinary Robinson's Patent Barley.

Dextrinized barley is rather indicated from the third month to the later periods of infancy, and even then this gruel is not as well borne by some infants as the ordinary barley-gruel above indicated. There is no question in my mind that the addition of a barley-gruel to a milk mixture aids in the assimilation of the curd of the milk. This can be well seen when an infant taking such a mixture spits up a small quantity after feeding. The curd thus rejected is very finely divided, and closely resembles the curd of mother's milk.

Dextrinized Gruels as Infant Food.

Jacobi was the first in this country to advocate the addition of a cereal decoction to milk in dilutions to aid the digestion of the casein in the cows' milk. From this has developed the addition of *dextrinized* gruels to cows' milk, with the same end in view. Chapin, in this country, and Keller, in Germany, are the prime movers in advocating for certain cases this method of infant-feeding.

So far as the Chapin method is concerned, it consists principally in dextrinizing a thin gruel of barley or flour by means of a diastase preparation (Cereo), adding this to the milk, and administering it in this fashion to the infant. Chapin advocates the administration of dextrinized gruels in combination with milk in percentage dilutions both for healthy and sick infants. The author cannot agree with him in this, and cannot see the necessity for dextrinizing any dilution of milk for the normal infant; although the author has found the method of adding decoctions of barley-gruel to the milk in percentage dilution one of the most useful methods of feeding infants with feeble digestion. He has never, however, proceeded to dextrinize a gruel and combine this with milk until he was convinced that no other method of feeding the infant would succeed. The author feels, therefore, that to dextrinize gruels in combination with cows' milk in every case is rather an extreme method of infant-feeding, and is uncalled for in a vast majority of cases.

Keller has advocated the use of these gruels with sick infants, especially of the marantic type, and in this respect the author's experience carries out the contention of Keller, that much can be obtained and brilliant results follow the use of these dextrinized gruels. The majority of pediatrists use no other diluent than water for the milk of normal infants, and in this respect the author's experience agrees with the majority. We shall therefore only elucidate this method of infant-feeding with dextrinized gruels as it applies to sick infants. In the present method some form of diastase, either pure or combined with malt extract, is added to the cereal dilution. Chapin takes a tablespoonful of flour, adds this to 1½ pints of water, and boils the mixture for fifteen minutes. He then adds a teaspoonful of a solution of diastase (so-called Cereo) to the mixture, the gruel becomes thin, and is then considered dextrinized. In this form it is added to the milk as a diluent in the requisite quantity. The author prefers the Keller method.

Keller utilizes the formula of Liebig in making a malt extract. To this malt extract potassium carbonate is added as an animal salt. One hundred grammes of this malt extract are added to 500 c.c. of water, or 1 pint, and dissolved. This is solution No. 1. He then suspends 50 grammes of wheat flour in 500 c.c. of milk, so that the solution is quite uniform. He then strains the milk and flour through cheesecloth. The solution of malt extract and that of the milk and flour are mixed together, put into a common vessel, and stirred constantly over a slow fire. After about twenty minutes of stirring the whole mixture is brought to a boil to stop all processes of digestion. The mixture is now put up in bottles, each containing about 6 ounces, corked, and kept cool. This mixture contains dextrinized cereal and malt-sugar in addition to the milk. The Liebig malt extract utilized by Keller is composed of maltose, 57 per cent.; dextrine, 12.4 per cent. Wheat contains 66.8 per cent. of starch, 7.5 per cent. of dextrine, and a small amount of dextrose. By the action of the ferments in the malt extracts—principally diastase—the starches are converted into sugars. By this method a number of easily assimilable substances are introduced into the economy. The action of these processes on the casein coagulation seems favorable to its assimilation. The Keller method of feeding with dextrinized gruel, carried out carefully, is one of the most useful methods of feeding marantic infants, and one in which in a great number of cases of atrophy has given me brilliant results. I have used this method of feeding in cases in which all other known methods have failed. These children had been fed on modified milk prescribed in a most careful manner by men who may be considered skilful in its administration. They had not thriven, and presented the picture known to most clinicians under the name of marasmus. Many of these infants were quite young, three months of age, and still, by careful administration of the Keller dextrinized gruel mixture in proper dilution, these infants throve, whereas other foods failed to produce the same result. Many of the infants who throve on the Keller dextrinized gruel had been subject either to chronic or subacute enteric catarrh. Keller has explained his success in feeding this class of cases by the fact that in them there exists an acid intoxication, emanating from the intestine, which is neutralized by this alkaline food. Ammonia is excreted in the urine in increased amount, thus indicating a form of disturbed intestinal metabolism. Under the administration of this food he found that the ammonia diminishes or disappears from the urine. Keller has given these infants malt extract without the cereal, but has failed to obtain the same results or any increase of weight.

I have found that the cases best adapted to the use of the Keller dextrinized gruel are the infants who remain stationary in weight, notwithstanding all attempts at percentage modification of the food, where simply cows' milk or cows' milk and cereal decoction alone have been used. Another set of cases are the pronounced cases of infantile atrophy, primary or accompanied with chronic enteric catarrh, in which the body-weight has run down and the infant is brought to us weighing 6 to 7 pounds, either too old for a wet-nurse, or in a station of life and amid such surroundings as to preclude the use of a wet-nurse. It must be said, however, that in feeding these marantic infants on dextrinized gruels, containing as they do thoroughly cooked food, and coming to us with a previous history of having been fed on either sterilized or cooked food for months, we must be exceedingly careful not to develop the symptoms of scurvy. When we have succeeded, therefore, in placing a marantic infant on a dextrinized gruel, and after two or three weeks the infant has begun to thrive, we should begin the administration of fruit-juices with the food, in order to prevent any scorbutic tendencies. It should be our object only in administering dextrinized gruels to increase the body-weight to that which would be normal, or nearly so, to the infant of the same age, and then gradually return to a milk diet, raw milk being preferred in these cases. Of course, when a return is made to a milk diet, it must be gradual, and we must be careful that the proper dilutions are made, else the old disturbances may return. As a matter of experience, we cannot keep these infants on the Keller dextrinized gruel beyond a period of three months. If kept longer on these gruels the children cease to increase in weight, remain stationary, and become anæmic; but if taken off in time the brilliant result continues. I have experienced no difficulty in a very large number of marantic infants in carrying out the above method successfully.

X. FOOD OF BREAST-FED OR BOTTLE-FED INFANTS AFTER THE SIXTH MONTH.

It has been shown by Camerer that the secretion of breast milk reaches its highest limits, both in quality and quantity, during the first six months of lactation. In many cases the quantity of milk diminishes, as also its quality. If the infant gains steadily in weight after the sixth month, no additional food is indicated. If, however, the increase of weight is not satisfactory, we may at this period begin with the daily administration of one or two bottles of modified cows' milk, in addition to the breast, continued until the infant is completely weaned. On the eruption of the incisor teeth, at the seventh month, the infant is allowed a cereal, in the shape of prepared barley, as a pap, with cracker or rusk of bread once or twice a day. If the infant is inclined to be constipated, the barley is omitted. The same procedure is followed as to cereals with bottle-fed infants after the seventh or eighth month.

XI. FEEDING FROM THE NINTH TO THE TWELFTH MONTH.

Breast-fed Infants.—Weaning.—It is not advisable to attempt weaning at the outset of the summer, even though we may be compelled to keep the infant at the breast a few months longer than usual. If the infant is partially weaned—that is, on a mixed feeding of breast and bottle—it should not be deprived of the breast entirely during the summer season. The reason for this is quite evident. During the summer a bottle-fed infant is very likely to be upset should anything happen to the milk. We would therefore be compelled to suspend the feeding with the bottle, proceed without milk for a few days, and then gradually return to the milk diet. In doing this our task will be less difficult if we have even a scantily secreted breast milk at our disposal. Convalescence from a dyspeptic attack will be much more rapid if return is made cautiously to breast milk than to a substitute feeding.

It takes about eight weeks to wean an infant completely. Sudden weaning of an infant from the breast is not only inadvisable, but in some cases attended with the greatest difficulties. If the infant has had the benefit of one or two additional bottles daily from the sixth month, the task of weaning is comparatively simple. If, however, the infant has been kept on the breast exclusively until the ninth month, when weaning is attempted certain difficulties will at once appear. The infant will not take the bottle if there is a breast at its disposal. The only way out of the difficulty is to deprive the infant at certain times of the day of the breast, and thus starve it into taking the bottle. This requires much moral courage on the part of the mother and of the physician. In those cases in which the mother nurses the infant we cannot always gain her co-operation in denying the breast to the infant. The difficulties of weaning in such cases are only increased, but with patience we can ultimately overcome them. I have seen infants who were deprived of the breast at this period refuse to take but a few ounces of nourishment daily for weeks. They emaciate, become restless, and refuse to be pacified. Under certain conditions, where the nursing function has been discontinued and the milk secretion has therefore ceased, the situation is at times really critical. But I have invariably seen the child take to his artificial food in due season, even if this surrender was delayed for a long period of time. Patience will ultimately conquer the little one in these cases.

In weaning I give those modifications of cows' milk which contain from 1 to 1.5 per cent. of proteids and 2 to 2.5 per cent. of fats until the infant is fully weaned. I then increase the strength of the milk to that given to the bottle-fed infant at the ninth month. At this time the bottle-fed infant is given almost whole milk. It is always well to mix with the milk a small quantity of water, in the proportion of 1 ounce of water to 7 of milk. Some infants who have been at the breast up to the ninth month will apparently refuse to take any modifications of milk which contain the cereal decoctions. In these cases I have tempted the infants with small quantities of raw milk slightly diluted with water, foregoing all attempts at percentage modification. This seems to have succeeded the best in trying cases.

In addition, the author gives from the ninth to the twelfth month, both to breast- and bottle-fed infants, cereals, in the shape of pap made up with barley, granum, rusk, and crackers, twice daily. For some of these infants an ounce of expressed beef-juice is mixed with equal portions of barley-water and slightly salted. This is given once a day. Infants relish this change.

XII. FEEDING FROM THE TWELFTH TO THE EIGHTEENTH MONTH.

At this period it is desirable to place the child on a diet containing a mixture of milk, cereals, eggs, and beef-juice, in the following manner: Five meals are given daily. At each, milk forms the basis of nourishment, generally accompanied by rusk or crackers. An egg is given once a day, perhaps beginning with the half and increasing to the whole egg as the infant grows older. At this time, also, fruit-juices, such as orange-juice, may be given to infants, especially to those who exhibit a rachitic tendency or who are constipated. The juice of half an orange daily will be relished by most infants.

XIII. SCHEDULE OF FEEDING FROM THE TWELFTH TO THE EIGHTEENTH MONTH.

Milk.—A quart and a half a pint to a pint daily.

Cereals.—Rusk or crackers, two of each a day; sponge cake in the form of long sugared slices; barley, granum, or oatmeal (the latter strained) in the form of a pap once a day.

Eggs.—One soft-boiled egg a day.

Meat.—Beef-juice expressed, mixed with equal portions of barley-water and slightly salted to the taste, about 2 to 4 ounces daily.

The above dietary is divided into five meals daily. If infants are markedly rachitic the author allows, in addition to the above dietary, a small amount of chicken meat, as much as will adhere to the bone of a chicken. This is given to the child once a day.

XIV. FEEDING FROM THE EIGHTEENTH MONTH TO THE END OF THE SECOND YEAR.

At this time the child is placed on a mixed carbohydrate and nitrogenous diet, consisting for the most part of milk, which is the basis of the diet; eggs; soup or beef-juice; meat of the beef or chicken; vegetables; cereals. These are divided, as in the previous period, into five meals daily, as follows:

Milk.—Some children will take considerable, some very little, milk at this period.

Eggs.—The eggs are boiled soft, as follows: A portion of water is brought to the boiling-point, taken from the stove, the egg placed in it, and allowed to remain for two minutes. Some children will take at least one egg a day, others two.

Soups.—Parents are apt to overstep the mark in giving large quantities of soup—in fact, an adult portion—to children. This is scarcely desirable, inasmuch at it displaces other food, such as milk, and, containing large quantities of salts and insoluble products, such as keratin, soup in large quantities is objectionable. The amount should not exceed 4 ounces.

Meats.—The ordinary boiled meat is by far the best for children. The inside of a lamb chop, a small piece of well-done beefsteak, roast beef, and chicken. Gamey meats, and fat meat, such as mutton, ham, pork, should be avoided.

Vegetables.—These include potatoes, peas, beans, carrots, spinach, the green vegetables being especially desirable, inasmuch as they contain iron. All vegetables should be given in a mashed form.

Cereals.—These should include barley, rice, granum, wheatena, oatmeal, rusk, crackers of all kinds, cocoa, and farina.

Fruits.—Orange-juice, ripe apples, and pears.

The articles of diet which should be avoided are vinegar, cabbage, salad, coffee, tea, wine, soups that contain too great an amount of amylacea.

A dietary consisting of the above foods might be formulated as follows:

Up to the end of the third year:

First Breakfast, 8 A. M.: 250 c.c. (8 ounces) of milk, with or without a cereal. A slice of bread or crackers and an egg.

Second Breakfast, 10:30 A. M.: 180 c.c. (6 ounces) of milk, 1 rusk, and the juice of half an orange.

Dinner, 1 P. M.: 120 grammes (4 ounces) of soup; 75 grammes (2.5 ounces) of meat with vegetables, and a fruit dessert.

Afternoon Lunch, 4 P. M.: 250 c.c. (8 ounces) of milk or cocoa with rusk or crackers.

Supper, 6:30 to 7 P. M.: Soft egg, 250 c.c. (8 ounces) of milk; cracker, toasted bread, or farina in the milk.

Candy.—I allow one or two pieces of candy daily, generally good chocolate, to older children.

From the third to the sixth year of life the diet should be mostly fluid or semifluid. The basis of all such diets should be milk. Milk soups, eggs, meat, butter, cocoa, bread, fresh vegetables, and fruits. The number of meals a day should be five.

The following is a schedule of a liberal diet at this time:

Breakfast, 8 A. M.: 330 c.c. (11 ounces) of milk, buttered bread, about half an ounce of sweet butter being allowed.

Dinner, 1 P. M.: 180 c.c. (6 ounces) of soup; meat, 90 grammes (3 ounces), vegetables, a dessert, generally of baked apples.

Afternoon Lunch, 4 P. M.: 240 c.c. (8 ounces) of milk, rusk or a slice of bread, or cracker.

Supper, 7 P. M.: 240 c.c. (8 ounces) of milk mixed with some cereal, generally farina, 1 soft egg.

This is a liberal diet. Some children will take as much as is here prescribed, others will take less. Some children are particularly fond of fish, and this may be given once or twice a week, generally in the boiled form with an egg sauce. Fried fish should never be allowed. It is advisable, especially in exceedingly nervous children or in those who have a lithic tendency, to substitute meat once or twice weekly by fish.

The above form of diet with slight modifications is suitable up to the tenth year of life. The object of all dietaries after the eighteenth month is to mix the carbohydrates, fat, and albuminoids in rational proportions. The following table by Camerer distinctly demonstrates this:

Age and weight	Second to fourth year. 12.7 kilos.	Five to six years. 18.7 kilos.	Seven to ten years. 24 kilos.
Total food (daily)	1183 grammes.	1517 grammes.	1699 grammes.
Albumin	46 "	64 "	67 "
Fat	39 "	46 "	32 "
Carbohydrates	117 "	197 "	251 "
Water	957 "	1200 "	1333 "

The Feeding of Sick Infants and Children.

The feeding of sick infants is considered under the headings of the various diseases. It must always be borne in mind that infants and children, if left to their own resources, would take either very little nourishment or too much. In certain marantic conditions infants will take very large quantities of food if it is given to them. The infant's cries are interpreted by the mother as being due to hunger, when they may be due to colic or intestinal distention. In these cases the mother gives too great a quantity of food, and the infants suffer from distention of the stomach. In typhoid fever, pneumonia, or other acute disease the patient, if fed at long intervals, takes but little food. I am in the habit of giving such infants or children small quantities at short intervals. If the infant takes a small quantity at each feeding, the aggregate amount in twenty-four hours is sufficient to maintain nutrition.

After operations, such as those for empyema, infants and children must be carefully and systematically fed up in order that they may combat the ravages of disease. The necessity of careful feeding is seen in typhoid fever in the fifth and sixth weeks, at which time there is great emaciation and the temperature has dropped to the normal. If we fail to feed up the patients, they remain emaciated and show slight inanition temperatures. On the other hand, we must not give large quantities of indigestible food. We must choose the foods carefully. Convalescents can take much larger quantities of food in twenty-four hours than the normal, healthy child. The quantity given at each feeding should be smaller than in health. The nitrogenous foods, such as milk and eggs, and also sugars, starches, and cereals of all kinds, are easily assimilable. Alcoholics, when given, should be well diluted. Rectal feeding is contraindicated in diarrhœal conditions and states of rectal intolerance. On the other hand, if the stomach rejects food repeatedly, it is well to give that organ complete rest. Under such conditions even water is not introduced into the stomach. The patient is fed for twenty-four hours or more by rectum.

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SECTION III.

DISEASES OF THE NEWBORN.

I. PHYSIOLOGY OF THE NEWBORN.

Respiration.

INASMUCH as cardiac action and muscular movement occur during the intra-uterine life of the fœtus, the first important function performed by the newborn infant is that of respiration. The cause of the first inspiratory act of the newborn has been a matter of much discussion; whereas some contend that mechanical stimulus brought into play by the act of parturition is the primary cause of the first inspiratory act of the infant, others have insisted that the change of temperature from the uterus to that of the external world, acting on the surface of the body, is sufficient stimulus to cause, by reflex action, the first act of inspiration. Both these theories have been disproved, especially by the work of Ahlfeld. The consensus of opinion is, that the first inspiration of the newborn is a direct result of the separation of the placenta with the cessation of the normal fœtal aëration of the blood; as a result of this there are diminution of oxygen in the fœtal blood, increase in carbonic-acid gas, and marked stimulation of the respiratory centre of the newborn in the medulla. This theory is borne out by the fact that in premature separation of the placenta this stimulus to the performance of inspiration on the part of the fœtus occurs before birth in the uterus or in any part of the parturient canal. There are rare cases also in which the fœtus is born before separation of the placenta from the walls of the uterus. In such instances the birth has been very rapid and the resistance to the passage of the fœtus slight; as a result of the uterus having contracted but little, the placenta remains *in situ* for a short space of time after birth of the fœtus. Such a case has been published by Kehrer, and in this case the fœtus was born in a state of uterine apnœa, the color of the skin being pink, the infant not breathing, but at most performing the intra-uterine respiratory movements of the trunk, as described by Ahlfeld. Such cases would seem to prove the truth of the theory that if the placenta still remains attached to the uterine walls, and the interchange of oxygen between the maternal and fœtel blood continues, the stimulus to the medullary centres mentioned above is lacking. There is thus no inspiration in such cases until the placenta separates. Ahlfeld has shown that *in utero* the fœtus performs certain rhythmic movements of the trunk and extremities, which he interprets as respiratory in their nature. There are thus, according to these experiments, respiratory movements performed by the fœtus *in utero*. These, however, are of the most superficial character, and do not lead to aspiration of liquor amnii either by mouth or nostrils. It is the intensification of these intra-uterine respiratory movements which eventuates in the first act of respiration of the newborn. Though the existence of intra-uterine fœtal respiratory waves as conducted by the liquor amnii to the uterine wall have been verified by a number of observers, their interpretation is diverse and their significance is still a matter of discussion.

The rhythm of respiration in the newborn is quite irregular. Deep inspiration and expiration are followed by regular respiration, with an apparent pause in which the respiratory movements are so superficial that the infant scarcely seems to breathe, and in which the respiratory movements can only be detected by the chymograph, this in a manner recalling the intra-uterine respiration of Ahlfeld. During sleep the respiration is more regular, but is influenced by the least external source of disturbance, such as a change in the surrounding light, air, and covering of the newborn.

The frequency of respiration can thus be of varying rapidity. Dohrn found that, regardless of sleep or waking, the number of respirations of the newborn was on the average 62; during the act of crying 47.

The type of respiration in the newborn, either in the male or female, is predominantly thoracic.

The aeration of the lungs—that is, the replacement of inspired by expired air—is much more thorough in the lungs of the newborn than later in life. In other words, if, as has been proved by Dohrn, 38 c.c. of air are inspired on the first day of life, this is renewed by each inspiration and expiration to such an extent that there is little residual air in the lung. In the adult lung the contrary obtains, there being, even on forced expiration, enough air left in the lung and retained there to be demonstrated by collapse of the organ when the thorax is opened and atmospheric pressure is allowed to act on the viscus. In the newborn, if the thorax is opened postmortem, no such collapse of the lung takes place, but enough air remains in the organ to enable it to float if placed in water—that is, a minimum amount of air. The lung of the newborn unfolds gradually, so that in an infant two weeks old there are patches of the lung which have still not been aërated, although the infant has breathed normally all this time. That the lung does unfold gradually is proved by the fact that, while immediately postpartum, 38 c.c. of air are taken into the lungs with each inspiration, this, on the tenth day of life, has increased to a volume of 50 c.c.

Circulation.

At the moment of birth of the infant certain changes take place in the circulatory system which mark the transition from intra-uterine as distinguished from extra-uterine life. These changes occur in the foramen ovale, the ductus Botalli, and the umbilical arteries and veins. On the first inspiration the lungs expand and the blood passes into the organ. The pressure is immediately lowered in the right auricle on account of the diminished resistance of the pulmonary capillaries; the pressure in the left auricle is correspondingly increased. The foramen ovale, situated in the auricular septum, with a valvular slit-like opening toward the left auricle, is naturally closed by the increased pressure in the left auricle, and from thence forward there is no interchange of blood between the right and left auricle as in fœtal life.

The closure of the ductus Botalli is a matter of much discussion. The explanation of its closure given by Strassman is now accepted by most observers. On expansion of the lungs and the establishment of the smaller pulmonary circulation, the pressure in the pulmonary artery is diminished and that in the aorta increased. The ductus Botalli, passing as it does from before backward from the pulmonary artery to the aorta, enters the latter vessel at an acute angle. Its lumen at the aortic extremity is funnel-like and closed by a slit-like valvular arrangement, whereas during fœtal life, the pressure being greater in the pulmonary artery than in the aorta, it was possible for blood to pass through the ductus Botalli into the aorta. At birth, the pressure conditions being reversed, it becomes impossible for blood to pass from the pulmonary artery to the aorta, the pressure in the pulmonary artery not being equal to driving the blood through the ductus against the increased postnatal pressure in the aorta. The ductus thus becomes emptied, and its function as a circulatory organ connecting the pulmonary artery and the aorta ceases. No clot is formed except in rare cases in the lumen of the ductus. *In utero*, from the fifth month on, there is a gradual diminution in the calibre of the vessel; within two or three days after birth the calibre is so narrowed that a probe cannot be insinuated within it. The aortic extremity in many cases is never entirely occluded, although most of the vessels become subsequently obliterated.

At birth the umbilical arteries are closed by a process similar to that which is described above. The first inspiration with consequent inflation of the lung and the establishment of the pulmonary circulation causes a fall of arterial pressure in the descending aorta. The blood ceases to flow through the umbilical arteries. The muscular coats of these vessels, being particularly well developed, tend to contract, and enclose in their lumen an extended fibrinous clot. When the cord is tied this clot extends from the umbilicus to the hypogastric arteries. It is the adherence of the thrombi to the walls of these vessels and their subsequent organization which causes the obliteration of the lumen of the umbilical arteries, although this is not complete except at the situation of the umbilicus. The umbilical veins are obliterated in a physiological manner by the pressure of the uterus on the placenta. This forces the reserve blood in the placenta into the body of the fœtus, the act of inspiration favoring the flow of blood from the placenta to the body of the fœtus. Budin has shown that if the umbilical cord is divided too soon after birth blood to the amount of about 100 c.c. flows from the veins. If ligation of the cord is delayed, however, this quantity of blood is aspirated, so to speak, by the infant into its body from the placental sinuses.

After ligation of the cord, therefore, the natural physiological condition of the circulation favors the collapse and obliteration of the umbilical veins.

Pulse.

The pulse of the newborn infant is irregular in frequency and shows certain constant characteristics. Immediately after birth the frequency reaches 150 to 190 beats a minute. This rapidity is due probably to the new conditions inaugurated at birth in the circulation and the increased amount of work, caused by respiration and pulmonary circulation, thrown upon the left ventricle. After a short lapse of time, from twenty minutes to an hour after birth, the pulse frequency sinks to less than 100 during sleep. During waking, nursing, crying, muscular exercise, there is a slight increase in frequency. After three to five days the pulse mounts in frequency from 120 to 135 beats per minute, but never again attains the rapidity observed immediately after birth. The rise in frequency of the pulse after its primary fall may be due not only to the recovery of the circulatory system, especially the heart, from the effects of rapid changes of tension in its various parts, incident to the new extra-uterine conditions, but to the beginning influence of the vagus on the left ventricle.

The influence of sex on the frequency of the pulse is evident in the newborn as in later life, the pulse of girls during sleep being two or three beats more frequent to the minute than that of boys. During exercise the pulse of the male newborn infant is more frequent than that of the female. The true cause of these differences is as yet obscure,

Blood.

The amount of blood of the newborn as compared with the body-weight varies with the time of ligation of the umbilical cord. If the cord is ligated at once the quantity of blood is one-fourteenth to one-sixteenth of that of the body-weight; whereas in cases of late ligation of the cord it is one-tenth to one-eleventh of the body-weight.

The histological characteristics of the blood of the newborn are so striking as to merit brief mention here. The blood contains a large number of nucleated red blood-cells; the red blood-cells do not tend to collect in rouleaux, and show very little or no central depression, as later on. The number of red blood-cells is not only greater to the cubic millimetre than later in infancy, but the hæmoglobin percentage of the blood and of the individual erythrocyte is much higher than later on. The red blood-cells show also the central "shadows" to a greater extent than is seen later in infancy. The white cells are present in larger numbers relative to the red blood-cells than later in infancy, and this proportion is still greater after feeding.

The white blood-cells have a marked tendency also to group themselves in clumps. These characteristics of the blood gradually disappear toward the eleventh day, and are most pronounced on the fourth day after birth. It can thus be seen that the blood picture obtained during the first days of life is such as would be of grave pathological import if found in the adult.

Digestive Functions.

The saliva is secreted in much less quantity in the newborn than later in infancy, and is present in just sufficient amount to moisten the mucous membrane of the mouth. Its reaction is slightly alkaline, but in disturbed conditions of the mucous membrane of the mouth it becomes acid. The amyllolitic ferments are present only in the secretion of the parotid gland, and here only to a slight degree. The secretion of the submaxillary gland shows this property only after the third month of infancy (Zweifel).

Pepsin is found in the stomach of the embryo at the fourth month, whereas *hydrochloric acid* is found in this organ only during the later months of fœtal life. Both are present in the stomach of the newborn infant.

The *pancreatic secretion* in the newborn, while incapable of converting starch into sugar, does contain trypsin and a fat-splitting ferment.

The properties of the secretions of the mucous membrane of the intestine of the newborn are still a matter of speculation. The gall-bladder of the newborn infant contains 0.1 to 0.3 grammes of bile, which is increased in amount after the ingestion of food. The bile contains less water and is richer in mucin, coloring-matter, and taurocholic acid in the newborn infant than at any other time of life. Glycocholic acid is not found in the bile of the newborn. The physiological function of the bile is still undetermined.

From the above it will be seen that in the newborn infant the digestion of starchy substances is but feeble, whereas the digestion of fats and albuminoids is as complete as in later infancy.

Body-temperature.

The rectal temperature of the infant taken immediately after birth is about 0.6° C. higher than that of the mother. The average temperature of the newborn subsequent to depressions incident to the immediate postnatal period is 37.7° C. (99.6° F.). Febrile states of the mother at the time of parturition, or external influences, may cause a rise or fall of the body-temperature in the newborn. Thus, a case is recorded in which an infant born of a mother suffering from fever at the time of labor had a temperature of 41° C. (105.8° F.) immediately after delivery (Lange, Fehling). Premature or congenitally weak infants have a lower rectal temperature than vigorous full-term infants. An hour or two after birth the body-temperature falls, but after nine to seventeen hours attains 37° C. (98.6° F.). This fall may be as much as 1.7° to 2.5° C., and is due to the cooling influence of the first bath, the change from the warmth of the uterine cavity to the external air, and the respiration. Toward the end of the first week the body-temperature of the newborn rises slowly to the permanent normal by tenths of a degree. In the congenitally weak the temperature rises more slowly and reaches 37° C. after twenty-four hours; whereas in strong and well-developed infants it reaches this limit in one-quarter to one-half this time. The temperature of the newborn is more easily depressed and raised by external influences than that of the adult. Thus, clothes and the surrounding atmosphere exert a marked influence in this respect. There is also a direct relation between the amount of nourishment ingested and the body-temperature. If the nutriment is insufficient, the normal temperature is attained much more slowly than under contrary conditions. An illustration of the influence of external conditions on the temperature is the case of a congenitally weak infant recently brought to my notice, whose temperature was raised fully 0.6° C. (1.5° F.) above the normal by placing warm-water bottles too near the body. The temperature of the newborn, therefore, is subject to wide variations; but it may be said that after the second day a temperature below 36° C. (96.8° F.) or above 38° C. (100.4° F.) is abnormal. The diurnal fluctuation of the body-temperature of the newborn is characteristic in that the highest point in the curve is reached in the early morning (6 A. M.) rather than in the evening, as in the adult.

Skin.

The body of the newborn infant is covered with a grayish, cheesy material, which consists of epithelial scales and secretions from the sebaceous glands, called the *vernix caseosa.* As is well known, this is washed off after birth, and leaves the skin smooth and of a uniform pink color. The skin of the newborn infant desquamates in small and large scales. This is distinctly noticeable at the sixth or seventh day, and ends in the second or third week after birth. Small vesicles are seen to form here and there on the skin over the body in some infants.

The body at birth is covered with soft, long hair called *lanugo.* This is also found on the scalp. In the first few weeks after birth this hair falls out and is replaced by the permanent hair. In weaklings this replacement, as also desquamation of the skin, takes place more slowly than in stronger infants. In the first few weeks the sebaceous glands are especially active, and their activity is indicated by the appearance of the so-called scurf on the scalp. On the body, in the groin, on the nose and face, small white bodies are noticed in the newborn infant, called *milia.* Epstein showed that these were really retention cysts of sebaceous follicles of the skin. They disappear in the course of two or three weeks.

Jaundice.—The skin, though pink in color at birth, becomes jaundiced from the second to the fourth day after birth in 80 per cent. of newborn infants.

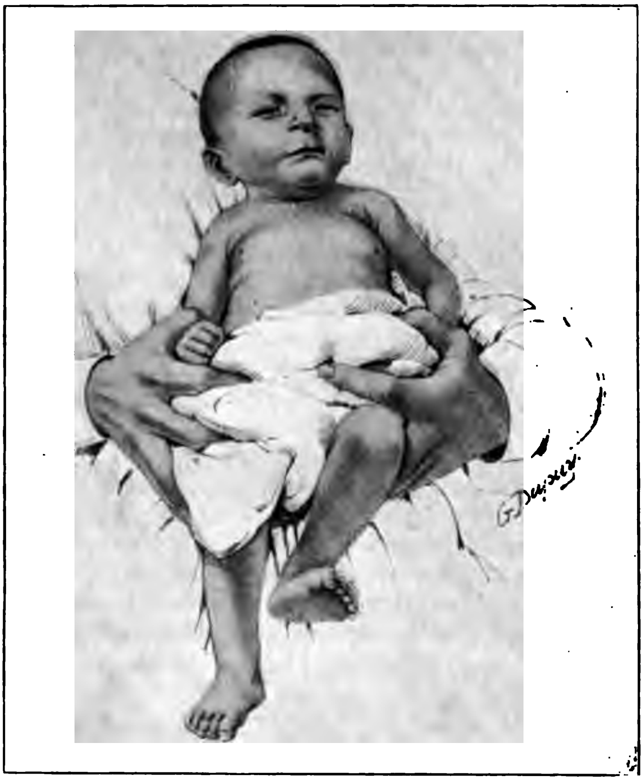
Perspiration.—Although infants are hardly seen to perspire profusely unless warmly clothed, the insensible perspiration from the skin and lungs is proportionately greater for the expanse of the body surface than in the adult. Rubner and Heubner showed that the infant yielded

During the first week fully 90 grammes (3 ounces);
" " second and third months 192 grammes (6½ ounces);
" " fifth and sixth " 290 grammes (9⅔ ounces);
" " first year 460 grammes (15⅓ ounces)
of insensible perspiration daily, as compared with 650 grammes (21⅔ ounces of the adult.

Breasts.

From the third to the fifth day after birth milk appears in the breasts of the newborn infant of both sexes. As a rule, the secretion appears earlier in the breasts of girls than in boys. The breasts become swollen and tense; one gland, generally the right, functionating sooner than the other. The cause of this curious phenomenon is as yet unknown. Balantyne suggests that it is due to a biochemical relation between the fœtus and the mother, which exercises its influence on the infant after birth in such a way that the same agencies which cause a production of milk in the mother continue to produce the same result in the infant. The secretion has been examined by Barfurth, Herz, and others, and has been found to be composed of proteids, 2.5 to 2.6 per cent.; fat, 2.3 to 3 per cent.; sugar, 2.5 per cent. It is therefore a real secretion of milk, and the method of its secretion is the same as in the adult gland. The amount of milk, which is called by the laity "witches' milk," is small. The secretion lasts, as a rule, from six to eight weeks; in exceptional cases it may continue six months (Herz). If mastitis occur, it is certainly the result either of antepartum or postpartum infection, and not of caking of the breasts.

FIG. 23.



Caking of the milk in both breasts of a newborn infant.

Urine.

Speaking of the urine of the newborn in a stricter sense, the amount passed spontaneously after birth is on the average 9.6 c.c., of which 7.5 c.c. may be found in the bladder at the time of birth, unless the viscus has been subjected to pressure during birth. The urine is passed spontaneously within twenty-four hours after birth in 66 per cent. of newborn infants, and in the remaining cases within forty-eight hours after birth.

The daily quantity of urine during the first two weeks varies widely according to different observers. On an average the amount varies in breast-fed and bottle-fed infants according to the amount of fluid food ingested. In breast-fed infants the amount during the first three days increases from 17 c.c. to 43 to 49 c.c., and on the fourth day amounts to 116 c.c., due to an increase of milk in the mother's breast. On the fourteenth day the amount has run up to 263 c.c.

Hofmeier and Schiff have shown that infants in whom the cord has been tied early, and in whom the gross amount of blood in circulation is less than in those in whom the cord has been tied late, the daily quantity of urine will be proportionately less. As soon as a constant relationship is established between the amount of fluid taken into the body and that excreted, toward the seventh day, then the amount of urine excreted reaches the proportionate relationship to the body-weight that exists in the adult. Thus, whereas on the first day 21.8 to 38.8 per cent. of the milk taken is excreted in the form of urine (Cruse, Reusing), on the eighth day it reaches the constant proportion of 62.8 per cent., as in the adult. The amount of urine proportionate to each kilogramme of body-weight must necessarily increase more markedly during the first eight days, inasmuch as, while the body-weight during this time is more or less stationary, the gross amount of urine increases. Thus, the first day it amounts to 5.9 c.c. to each kilogramme; on the eighth day it reaches 67.4 c.c., on the tenth day 90 c.c., and then remains stationary. These figures are higher than is true of the adult, in whom the daily amount of urine per kilogramme of body-weight is 25 c.c.

The urine of the newborn is almost colorless; its specific gravity in the fœtus 1002.8 (Dohrn); in the first two days of life 1008 to 1009, on the third day 1011 to 1013, and on the tenth day it falls to 1003 to 1004. In bottle-fed infants the specific gravity is lower than in the breast-fed infant, due to the increased amount of milk ingested. The reaction of the urine of the newborn is constantly acid in all but 3 per cent. (Hofmeier). The urine of newborn infants during the first five days of life contains renal epithelium, uric-acid crystals, amorphous urates, and frequently casts. Hyaline and granular casts were found by Reusing in 39.4 per cent. of breast-fed and in 9 per cent. of bottle-fed infants during the first days of life. If casts are present, there is also, as a rule, albumin.

The amount of urea increases absolutely and relatively from the first day of life. Thus, it increases in absolute quantity from 0.06 to 0.11 the first day, to 0.82 on the eleventh day, or, relatively to each kilogramme of body-weight, 0.018 to 0.29 on the seventh day.

Uric acid is present in the urine of the newborn in remarkably large amounts. Thus, on the fist day the urine contains 0.0136 of uric acid to each kilogramme of body-weight. That is much greater than in the aflult. The proportion of uric acid to urea is much greater in the newborn than at any other period of life; in the adult it reaches the high relative percentage of the newborn only in pathological states. Thus, in the adult the relative proportion of uric acid to urea is as 1 : 41 or 61, whereas in the newborn it ranges from 1 : 1.5 on the first day to 1 : 21.9 on the seventh day of life.

Albumin is found immediately after birth in the urine in 38 per cent. of infants (Dohrn). Dohrn ascribes its presence as due to complications during birth or disturbances, however slight, of the circulation. Hofmeier found it disappeared toward the end of the first week; also that there was a constant relationship between the excretion of uric acid and the presence of albumin in the urine of the newborn, the latter being absent in those cases in which uric acid was not found and in which no uric acid infarction of the kidney existed. He attributed the albuminuria of the newborn to the mechanical irritation of the deposits of uric acid on the epithelium of the uriniferous tubules.

Rectal Excreta.

Meconium forms the gut content in the fœtus. It is of a yellowish-green color in the small intestines—of a dark-green color in the large gut, becoming lighter after an interval of a day or so after birth. It is of a tarry consistence and odorless. The total quantity of meconium varies from 70 to 90 grammes, or 2½ to 3 ounces, of which 2 to 20 grammes are passed daily. When the infant takes the breast or cows' milk, meconium is mingled in the movements with the milk fæces. The first passage of meconium occurs immediately after or in the first few hours, at the latest ten to twelve hours, after birth, and is preceded by the expulsion of the so-called meconium plug. This is a body of mucoid tissue 2 millimetres in diameter, and is of importance in a medico-legal sense. An infant stillborn will retain in the lower part of the rectum the meconium plug.

The stools for the first two days consist mostly of meconium, which subsequently becomes mingled with the milk fæces. The movements contain both yellow and greenish residue. After the fourth day the infantile movements assume their permanent characteristics of color and consistence. The composition of meconium has been fully investigated. It is made up of desquamated epidermal and intestinal epithelium, amniotic fluid, vernix caseosa, wool-hair or lanugo, plates of cholesterin, scales of skin, hæmatoidin crystals, bilirubin, fat-drops, and stearic-acid crystals. Bilirubin is peculiar to meconium (Zweifel and Schmidt). Weintraud found uric acid and alloxur bases in meconium, which were probably derived from a nuclein substance. Schild found that sterile meconium contained a peptonizing ferment; and Patevin found a lab-ferment and amylase in sterile meconium.

Meconium contains also the characteristic so-called "meconium bodies." These consist of ovoid or polyhedral masses yellowish-green in color. They are made up of organic matter, such as masses of intestinal epithelium and mucus, in which are precipitated biliary pigments and salts. They contain biliary pigment, soluble in caustic potash, insoluble in ether or acetic acid.

Chemical analysis of meconium reveals mucin, palmitin, stearin, olein, biliary pigments, and taurocholic acid. It does not contain indol or phenol, which are products of decomposition.

During the first few days of infancy the stools contain much mucus of a stringy character, and the writer has frequently seen this actually drawn out of the rectum in shreds by the nurse in otherwise normal infants.

Bacteria.—Meconium is sterile at first, and then becomes infected with bacteria in from three to eighteen hours after birth. A proteus similar to that of Hauser's is regularly found, also a chain coccus and a Bacillus subtilis. With the appearance of the milk fæces, a bacillus similar to that of the Bacillus lactis aërogenes is found in the upper part of the gut, the colon bacillus in the lower portion with the coccobacillus of Fischl.

Nervous System.

The nervous system is not in an active, but rather in a receptive, state in the newborn.

Muscular power as well as muscular sense is but little developed. The newborn infant can neither sit up nor hold its head upright. The reflex irritability of nerve and muscle both to galvanic or faradic stimulus is less evident in the newborn than later in infancy or in the adult. Response to stimulation is distinctly delayed; the latent period is more marked in the newborn. In the newborn the inhibitory functions of the vagus are not fully developed, but it is susceptible to reflex action, as is demonstrated in cases of cerebral pressure with slowed pulse due to injury incident to birth. In these cases the vagus would seem to exert through the cerebral centres an inhibitory influence on the heart. The cerebrum seems to be in a passive rather than in an active state in the newborn. In spite of the divergence of views, there seems to be no sign of consciousness in the newborn, nor are the motor centres developed to such an extent as to react under stimulus. Motion is rather of a reflex nature or indirectly referable to the high development of the sense of touch. Thus, we meet with injuries of the skull-cap in the newborn which are of an extensive character, such as depression of the skull, giving no symptoms referable to the motor areas. On the other hand, there is sufficient reason to believe that the cerebrum exerts a negative inhibitory influence, and that several of such centres are active in the newborn. The skin reflex presents nothing peculiar in the newborn. The patellar reflex is somewhat increased, diminishing after the seventh to the nineteenth day.

In spite of the assertions of Kussmaul and Preyer as to the existence of the sense of taste in the newborn, there is reason to believe that this sense is but little developed, and really exists in the nature of a reflex rather than a sense which distinguishes between sweet, bitter, and sour, as in the adult (Gensmer). Thus, Lange has given a 4 per cent. quinine solution to the newborn without awakening any signs as to the appreciation of its bitter taste. It is also questionable whether the newborn appreciates the sweet taste of breast milk. On the whole, it may be said that it takes a strong solution of any kind, sweet or bitter, to cause any visible reaction in the newborn, and that this reaction is rather in the nature of a general reflex than an appreciation of differences of sweet, sour, or bitter.

Hearing is not evident as a sense immediately after birth, and, as has been pointed out elsewhere, the newborn infant is deaf. The sense of hearing develops at various periods after birth, from six to forty-eight hours, according to the rapidity with which in the newborn the Eustachian tube is opened up and air enters the internal ear. In prematurely born infants, on account of marked swelling of the walls of the tube, the development of the sense of hearing is much delayed. These facts explain the wide difference among observers (Kussmaul, Preyer, Gensmer) as to the development of this sense. Gensmer is probably correct when he says that most infants react to sound after the first or, at least, the second day. The improvement in the hearing is unmistakable in the first week.

In the premature as well as in infants born at full term, the eye reflexes are developed. Thus the pupil contracts and dilates under stimulus, and intense light or continued flashing of light in the vicinity of the newborn calls forth signs of general reflex irritability; that is, the newborn becomes uneasy under irritation of this nature. It is still a matter of discussion as to whether the newborn can fix or focus objects and whether accommodation is developed. The eyelids react promptly to reflex stimulus.

The sense of smell is but slightly developed in the newborn, and it is a matter of question as to whether at this time the infant may recognize the mother or the nipple of the breast by means of this sense. Far more probable is it that in this respect the sense of touch and its reflexes has been mistaken in its manifestations for that of smell.

The sense of touch is the most highly developed sense in the newborn, and is most evident in the lips and face. The lip reflex is especially developed, inasmuch as in the newborn the least contact of any object with the lips calls forth the pursing of the lips and the motions attendant on suckling.

The appreciation of pain is absent immediately after birth, and only after one or two days does the newborn react to the irritation of a pin-point.

But little is known of the appreciation of heat and cold in the newborn; and it may be said that these call forth only manifestations of a general reflex action as is seen in cases of asphyxia when infants are brought rapidly from the warm to the cold plunge.

Metabolism.

Though much is still to be learned as to the processes of metabolism in the newborn, there are certain facts as to the daily quantity of milk taken, the amount of urine voided, the loss of weight by means of the skin and fæces, which have been determined within certain limits.

The *amount of milk* consumed daily by the newborn has been carefully determined by weighing the infant before and after nursing. A well-developed infant nursing a normally secreting breast will, according to the investigations of Cammerer, Hähner, and Laure, consume the following quantities of milk, expressed in cubic centimetres:

Days. .	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
	33	123	209	290	305	342	400	417	426	413	441	437	516	487	536

Excretion and Waste.

Meconium and Fæces.—As long as meconium is voided (60. to 90. grammes) the movements are small. As soon as milk fæces appear they average 1 to 3 grammes of fæces to 100 c.c. of milk ingested. An infant during the first two weeks rarely voids more than 10 grammes of fæces daily. The excretion of carbonic-acid gas and water by the skin and lungs has as yet not been accurately determined. The experiments of Förster as to the excretion of carbonic-acid gas were performed on a fourteen-day-old infant. His observations were made on the sleeping infant only, also a source of error. Cammerer, however, determined the daily exhalation of carbonic-acid gas more definitely, and found that this was as follows:

1st day.	2d day.	3d day.	End 1st week.	End 2d week.
100 grammes	85 grammes	80 grammes	100 grammes	130 to 150 grammes

It may be said that under similar conditions the newborn infant exhales more carbonic-acid gas per kilogramme of body-weight than the adult.

Though certain facts as to the metabolic processes are as yet undetermined in the newborn, we can still form an approximate estimate as to the ultimate disposition of the food and the manner in which oxidation processes of the body are carried out in the first few days of life. Thus, Cammerer has by estimating the amount of food ingested, and the amount of urine, fæces, and carbonic-acid gas excreted, drawn up a very instructive table showing the loss of weight and the manner in which it is kept within certain limits during the first four days of life:

Day.	Milk taken.	Excreted.		Loss of weight.
1	10.0	Urine . . . . Meconium . . . . . . Co_2	48.0 51.0 96.0 195.0	185
2	91.5	Urine . . . . Meconium . . . . . . Fæces . . . . Co_2	53.0 23.0 3.0 84.5 163.5	72
3	247.0	Urine . . . . Fæces . . . . Co_2	172.0 3.0 82.0 257.0	10
4	337.0	Urine . . . . Fæces . . . . Co_2	226.0 2.5 93.5 322.0	+15

Thus, the loss of weight during the first four days is due in great part to the lack of sufficient nourishment to compensate for the loss through the urine and fæces.

In order to illustrate more completely the oxidation processes in the body, Cammerer has reduced the food and excreta to their chemical elements. The C. H. N. and O. taken into the body in the form of nourishment and inhaled oxygen are compared to the same elements excreted in the urine, fæces, and the expired air. It is seen that in the newborn, as in the adult, fully 93.4 per cent of the carbon taken in is excreted in the carbonic-acid gas, the nitrogen being excreted for the most part in the urine. Whereas, however, in the adult the nitrogen taken in is excreted entirely in the urine and fæces; in the newborn fully half the nitrogen is retained in the body.

Infant Fourteen Days Old, Weighing 3500 Grammes.

Taken into the body in twenty-four hours.		Water.	C.	H.	N.	O.	Ash.
Milk.	500.0	444.0	29.5	4.5	2.0	18.4	1.6
Inspired O.	70.2	. .	. .		. .	70.2	
		444.0	=	49.3		394.7	
	570.2	444.0	29.5	53.8	2.0	483.3	1.6
Urine voided . . .	350.0	347.0	0.7	0.1	1.0	0.7	0.5
Fæces	7.0	5.0	0.8	0.1	0.1	0.3	0.7
Co_2	185.1	104.1	22.0	. .	. .	59.0	
		456.1	=	50.7		405.4	
	542.1	456.1	23.5	50.9	1.1	465.4	1.2
Balance in favor of increase of weight	. .	28.1	6.0	2.9	0.9	17.9	0.4

The student may thus see that the loss of weight during the first few days is considerable. It has not as yet been accurately determined whether an infant nursed from the first day of life on a breast secreting abundant milk would lose weight similar to that of the newborn nursed on the mother's breast. It is well known that the loss of weight is greater in those fed on a substitute than on the breast. The further details of loss and increase of weight will be found under the heading of Infant-feeding.

II. MORTALITY AND SUDDEN DEATH IN THE NEWBORN.

Sudden death is not uncommon in the first week of life, and, according to Snow, fully one-tenth of the race succumb in the first month of existence. Modern methods have tended to reduce this startling mortality, but the conditions attending the birth of the infant are such that there will always be, independent of sepsis of any kind, a quota of the newborn which will succumb, either with previous symptoms or suddenly, in the first week or first month of existence. Eröss has found that 9½ per cent. of all children born in Europe died in the first four weeks of life. Of these 37 per cent.

died in the first week, 29 per cent. in the second, 21 per cent. in the third, and 13 per cent. in the fourth week.

The statistics of the different countries vary, as one would naturally expect, according to the methods of midwifery in vogue. It is of interest, however, that, in the United States, Snow, of Buffalo, has collected some statistics on this subject in his own city. Of 7290 births 471 died during the first month. Thus 6.4 per cent. of all the children born in Buffalo died during the first four weeks, a death rate of 9.3 per cent. of the total mortality.

If we look for the causes of mortality in early life we may class them grossly under those due to (1) immaturity or congenital weakness, with or without syphilis; (2) malformations which are fatal in themselves; (3) asphyxiation and atelectasis; (4) injuries sustained during parturition, such as apoplexies, both cephalic and abdominal; (5) septic infections of various kinds. The effect of prolonged and difficult labor, abnormal presentations, the application of forceps, may cause a cerebral hemorrhage, especially in premature or congenitally weak infants, but a difficult labor is not necessarily an etiological factor in these cases, for cerebral hemorrhages occur in infants who have passed through an apparently normal, or even a precipitate, labor. It seems that in these cases simple pressure of the parts in the parturient canal precipitates a hemorrhage which, subsequent to birth, attains an extent which is fatal.

Spencer found that of 130 infants dying in the first few hours of life, 65 per cent. of deaths were due to injuries sustained by the brain in the form of congestion and hemorrhages, and he considers the forceps, next to abnormal presentations, such as foot and breech, as the most frequent etiological factor in producing hemorrhage.

Hemorrhage and apoplexies of fatal character may occur in the liver, suprarenal capsule, and lung, and many children subject to cerebral hemorrhages succumb to convulsions in the first hours of existence. It must not be forgotten, however, that the most trying cases of sudden death in the newborn are those in which infants have been born after a labor in every respect normal, and who at birth presented absolutely nothing abnormal physically to the careful and practised eye, and who have continued in apparent health for twenty-four hours to a week, with sudden death as an outcome. These cases present absolutely no symptoms to warn the physician of the approaching catastrophe. They may nurse in a regular manner, apparently, the bowels may appear natural in color and consistence, and even after the death of the infant an inquiry into the clinical history of the case fails to reveal any symptom which might have led to the detection of the trouble. These cases postmortem may reveal a cerebral hemorrhage or an abdominal umbilical hemorrhage, which previously revealed but few symptoms. I recently saw such a case in a premature child, born rather precipitately, which continued well and in perfect condition for twenty-four hours, then suddenly developed cyanosis, attacks of respiratory apnœa, and died within a few hours. In this case nothing was revealed postmortem but a slight atelectasis of the lung. Another, in which an infant nursed on the breast of a wet-nurse for six days, did not lose weight, but rather held its own, nursed vigorously a few hours before death, cried but little, slept most of the time, and was found dead in bed, with a slight hemorrhage from the nose, on the sixth day. This, in all likelihood, was a case of unrecognized sepsis of the newborn.

In the newborn all cases do not die suddenly. There is in most cases marked or slight warning, extending over days or hours before the fatal issue. There may be signs of cerebral irritation, but these, as a rule, come on suddenly in severe cases. Some time, hours or days, after birth, the child may be attacked with cyanosis, it may whine or cry without apparent cause, there may be derangement of the respirations, irregularly slow pulse; there may be a series of convulsions, which may end the scene or which may continue for days. Sometimes a slight hemorrhage gives rise to no symptoms at all until later in life, so that we cannot say that hemorrhage always causes death in the newborn, for this is contrary to experience.

If meningeal hemorrhage is preceded by the symptoms such as have been detailed, a diagnosis can be made, but in cases in which these symptoms are absent clinical diagnosis is impossible. Asphyxia, atelectasis, and compression of the cord cause a mortality of 3.6 per cent. of the total number of deaths in the newborn. In congenital atelectasis there may have been an easy labor, but inherent weakness and immaturity of the respiratory muscles may cause imperfect expansion of the lungs.

A large proportion of deaths in the newborn infant are the result of sepsis. The pathogeny and symptomatology of sepsis will be considered under the proper heading, but some of the severest forms of sepsis, resulting in arteritis of the umbilical arteries or in a general bacterial invasion, give absolutely no symptoms and result in sudden death. The conditions at this time of life are particularly favorable, as has been repeatedly pointed out in these pages, to the invasion of germs, and the avenues of infection are various, as has been dilated upon in the chapter on Sepsis in the Newborn. Not only is the resistance almost *nil*, but the progression of the disease is unhampered by such conditions as leucocytosis, which obtain later in life, for lack of leucocytic reaction and deficient development of the lymphatic apparatus is especially characteristic of this period of life.

In addition to sepsis, sudden death in the newborn may be due to forms of respiratory disease, such as bronchitis and pneumonia, which have not only escaped observation, but which give absolutely no symptoms before the fatal issue supervenes, and are only revealed on the postmortem table. Such infections have been dilated upon elsewhere. They may originate in foul atmosphere, unclean bedding, aspiration of amniotic fluid, and as a result of this, contamination by colon bacillus, staphylococci, and streptococci. We will not enlarge upon the other forms of sepsis of the gastro-enteric type, but will leave that for future consideration in the chapter on Sepsis. Sudden death in the newborn, due to hypertrophy of the thymus, is a great rarity.

III. CONGENITAL ANOMALIES.

Anomalies of the Scrotum.

The scrotum may be divided into halves, separated completely from each other, each with its contained testis. There are rarely more than two testes. There may be accompanying abnormalities, such as circumscribed hydrocele of the cord, lipoma fibrosum, and omental structures. Anorchidie is a condition of rudimentary or lacking testis and adnexa mostly unilateral. Ectopia testis abdominalis is a condition in which the testis is found underneath the skin of the abdomen. Ectopia cruralis testis is a condition in which the testis is found at the femoral ring, generally with a hernia. Ectopia perinealis testis is a condition in which the testis is found in the perineum.

Retentio Testis:

Retentio testis refers to those cases in which the testis remains in the abdominal cavity or in some part of the inguinal canal.

Retentio abdominalis refers to the retention of the testis in the abdomen.

Retentio iliaca, near the internal ring.

Retentio inguinalis refers to the testis retained in the canal or near the external ring.

Double retention is also called cryptorchism ; single retention is spoken of as momorchism.

These congenital conditions are quite common in children, but disappear, as a rule, toward the age of puberty. The cause of the congenital anomalies is a lack of development or peritoneal adhesions, and their principal interest clinically lies in the fact that they may be confounded with hernia of an inguinal type. The retained testis of the inguinal variety is apt also to atrophy, inasmuch as it is easily exposed to traumatism. Kocher has shown, also, that it is more apt to be the seat of new growths, especially carcinoma.

The diagnosis of retained testis, especially of the inguinal variety, is not difficult. The mother will invariably call attention to the absence of the testis from its usual situation. Examination of the scrotum will reveal its absence either on one or both sides. By invaginating the scrotum through the inguinal canal, the physician, as a rule, will find the testis in some part of the canal or at the internal opening in the abdomen as a small globular body. Tracing the location of the testis, its absence from the scrotum and its presence in the abnormal position mentioned, differentiates it from a lymph node or a hernia. As a rule, hernia as a result of coughing or exertion, such as crying, will descend and increase in size or protrude from the external ring. Not so with the testis. It may even retract higher if pain is experienced.

Treatment.—There is no treatment for this condition, although the French advise the systematic pushing down of the testis into the scrotum at certain intervals up to the age of puberty.

Hydrocele Congenita or Adnata:

This anomaly of the congenital type is caused by a lack of closure of the peritoneal fold, the pars vaginalis peritonei. There is a communication of the cavity of the tunica vaginalis with the peritoneal cavity to a greater or less extent. Serous fluid of the peritoneal cavity may gravitate to the cavity of the tunica vaginalis; or there may be a free opening into the peritoneal cavity, allowing a reposition of the fluid. In such cases the anomaly is apt to be confounded with inguinal hernia. The communication with the peritoneal cavity may be of filiform size.

The diagnosis from hernia is made possible by the fact that in the latter reposition with intestinal gurgle is possible; whereas a hydrocele cannot be reduced unless there is an opening through to the peritoneal cavity. On gentle percussion a hernia will also give tympany. Hernia will increase in size as a result of coughing or crying. Congenital hydrocele may disappear spontaneously. Irregular adhesions in the canal may result in small collections of fluid along the course of the spermatic cord, thus forming hydrocele of the cord. When there is a communication of the peritoneum with the tunica vaginalis, a large hernia may result.

The diagnosis of hydrocele of the cord in the young infant is often required of the physician. In these cases we find a collection of fluid around the cord in its course from the peritoneum to the testis. This fluid, however, does not communicate with the cavity of the tunica vaginalis testis; nor can the fluid be replaced, as the hernia can, into the abdominal cavity. The fluctuating swelling extends from the testis to the external abdominal ring.

Treatment.—The treatment for congenital hydrocele or hydrocele of the cord is that of repeated puncture and withdrawal of the fluid.

No irritants of any kind should be used in congenital forms of hydrocele, inasmuch as peritonitis may result should any anomalous opening into the peritoneal cavity exist.

IV. THE CONGENITALLY WEAK (Premature Infants).

Infants are congenitally weak who weigh less than 2000 grammes (4½ pounds), have a body-length of 42 centimetres, and who, on account of a lack of development of the various organs and a consequent imperfect performance of their functions, show a diminished vital energy. Such infants may be premature, weigh as little as 600 grammes (1⅓ pounds), with a body-length of 21 centimetres, and still live. As a rule, however, any infant weighing less than 1000 grammes (2.2 pounds) cannot live. The temperature must also be considered in the study of the congenitally weak, as well as the body-weight, for not only does this factor influence the prognosis, but also the management of these cases. Congenital weakness may thus exist to various degrees.

Etiology.—Prematurity is a most frequent cause of congenital weakness. The early interruption of pregnancy may occur in apparently healthy mothers as a result of mechanical influences, intercurrent infectious disease, diseases of the placenta or uterus, or constitutional disease. Congenital weakness may exist in one of twins or triplets, the other infants being born strong and well developed. Though most frequently found among premature infants, congenital weakness may exist in infants born at full term, as a result of the debilitating influences of tuberculosis or syphilis in the mother on the development of the fœtus. The congenitally weak are also found among infants who are born at full term, but in whom there has been for some reason no complete expansion of the lungs and in whom atelectasis results (asphyxia of the newborn). Thus, congenital weakness may at times go hand in hand with prematurity; at others prematurity is not an essential factor.

Morbid Anatomy.—Premature infants are underweight according to the degree of prematurity. The head is small and globular; the pupils still show the pupillary membrane; the skin is red and glistens; the face is wrinkled; wool-hair or lanugo covers the body; nails are undeveloped; the external genital organs, the clitoris and nymphæ, are prominent; the brain is undeveloped; the heart and vessels present fœtal characteristics, such as an open ductus Botalli or foramen ovale; the thyroid and thymus glands and the suprarenal capsules are large; uric-acid infarctions are found in the kidney; the intestinal structures and bones are undeveloped.

The lungs show areas of bronchopneumonia with atelectasis; on the surface of the lungs there are hemorrhagic areas resembling infarctions. In other words, there is hemorrhagic pneumonia due to infection either by streptococci, staphylococci, bacillis coli communis, or pneumococci. The bronchial nodes may be enlarged and there may be pericarditis. The intestines, liver, and kidney, in addition to being undeveloped, may present lesions similar to those found in sepsis. Infections may remain local and limited to the point of entrance of the bacteria, or may become general. The portals of infection are solutions of continuity in the skin, mucous membrane of the gastro-enteric tract and respiratory passages which allow the entrance of bacteria from the air, garments, or objects brought in contact with the infant's hands, linen, or food.

Symptoms.—The body is spare; the skin is soft and delicate, uniformly red and transparent, showing plainly the bloodvessels. The delicacy of the skin renders it susceptible to traumatism, resulting in the formation of erosions. The surface is cool, pale, icteric, sometimes cyanotic. Desquamation of the skin, present normally in the newborn, is delayed from four to eight weeks. In very severe cases there may be sclerema.

The infant does not cry, but rather whimpers; the respiratory movements are scarcely noticeable, there is muscular inertia, and the infant lies in a torpid condition. The intestine and stomach are easily disturbed; the liver performs its function imperfectly, and in many of these cases there is icterus. A temperature as low as 30° C. (86° F.) may exist and continue for days. These infants, if left exposed momentarily, even after a warm bath, may experience a serious reduction of temperature. They are thus easily chilled, and attain a temperature near the normal only with the greatest difficulty. The body-temperature during treatment in the incubator may not rise above 36.9° C. (98.4° F.). In those infants affected with sclerema the temperature may not rise for days above 28° to 35° C. (82.4—95° F.) in the rectum. As a direct result of the low body-temperature and disturbed metabolic processes these infants suffer from cyanosis, which at times is difficult to dissipate.

There is at first a lack of nursing power, and at most 10 or 15 c.c. of milk are taken at a nursing. The evacuation of the bowels takes place very sluggishly, often days apart; meconium persists in the gut as long as six to eight days. The urine is passed in much diminished quantity, and the loss of weight is more rapid than is true of normal infants.

Should a premature infant develop an infectious bronchopneumonia, the diagnosis is extremely difficult. Percussion can rarely establish a dulness of any extent, the respiratory movements are feeble, the air scarcely enters the lungs, cyanosis is present, and the temperature may be subnormal. The infant will therefore simply fail in a general way. There may be an eruption or hemorrhages in the skin, and death may take place with general or partial convulsions.

The congenitally weak infant may, if fed incorrectly, either with too much or faulty food (milk), suffer from diarrhœa, which tends not only to a reduction of body-weight, but to an increase of weakness.

Prognosis.—The body-weight, the rectal temperature, and the mode of feeding determine the prognosis.

Of the congenitally weak weighing less than 1200 grammes, but few or none are saved; of those weighing 1200 to 1400 grammes, 40 per cent. are saved; of those weighing 1500 to 1599 grammes, 86.7 per cent. are saved; and from 2000 to 2500 grammes, 93.6 per cent. are saved (Budin).

As an exceptional instance of successful rearing of the congenitally weak may be mentioned the case of Villemin, who records the saving of an infant who at birth weighed only 955 grammes (2 pounds).

The influence which the rectal temperature has on the prognosis is shown by Budin, who found that of cases weighing less than 1500 grammes, with a rectal temperature of 32° C. or less, only 2 of 103 were saved; of those weighing 1500 to 2000 grammes, with a rectal temperature of 32° C., only 1 of 39 was saved: a combined mortality of 98 per cent. Therefore the rapid reduction of temperature is an important factor in the mortality of these infants.

The mode of feeding is an important element in the prognosis, for the mortality is greater among the congenitally weak or premature infants brought up on the bottle than among those reared on the breast.

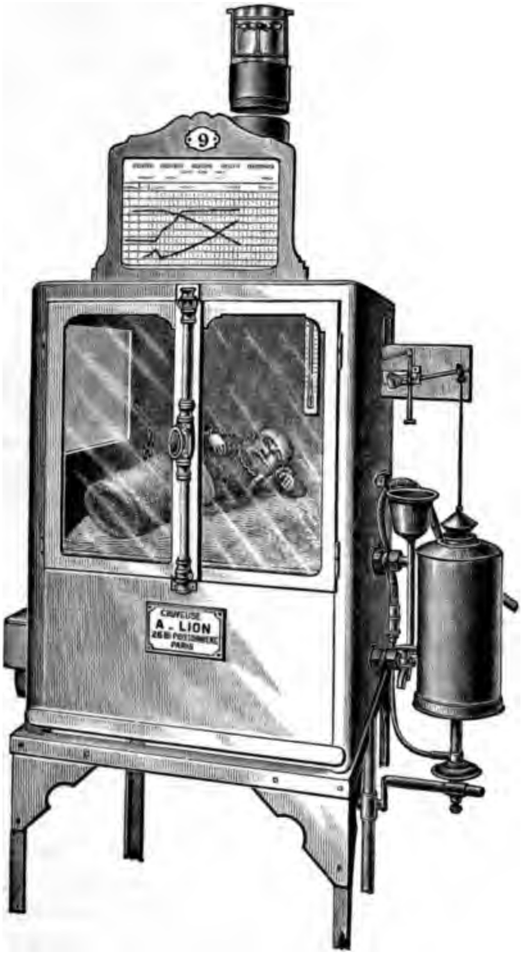
It is interesting to note the observation of Budin, that of 54 infants who had at departure from his service weighed 2800 to 3000 grammes, 31 per cent. died. Of the 54 infants, 24 were fed artificially, of whom 41 per cent. died; of 20 fed at the breast, only 15 per cent. died.

The causes of death among the congenitally weak, when brought up by methods to be described hereafter, are principally infectious bronchitis, bronchopneumonia, infectious and epidemic disease. Aside from infectious diarrhœa, syphilis and digestive disorders play an important rôle as causes of death.

Management of Congenitally Weak Infants.

In speaking of the management of congenitally weak infants the student should understand that each country has its favorite method of managing these cases; and it may be stated at the outset that we may well copy the methods of the French, who have made a very close study of the art of saving the premature and congenitally weak. If a premature or congenitally weak infant is born asphyxiated, the treatment is much the same at the start as that detailed in the section on asphyxia of the newborn; but, as intimated, our efforts must be directed to saving the congenitally weak, after resuscitation methods have succeeded, by maintaining the body-temperature, by feeding the infant correctly, and by supporting the heart and respiration. The weight and the rectal temperature, therefore, not the age of the infant at birth, will decide for the most part the line of treatment, for some infants at full term, as has been stated, are much below the normal weight, with a subnormal temperature, and are therefore congenitally weak. It would be unsafe to outline any treatment based only on the age of the infant at birth.

FIG. 24.



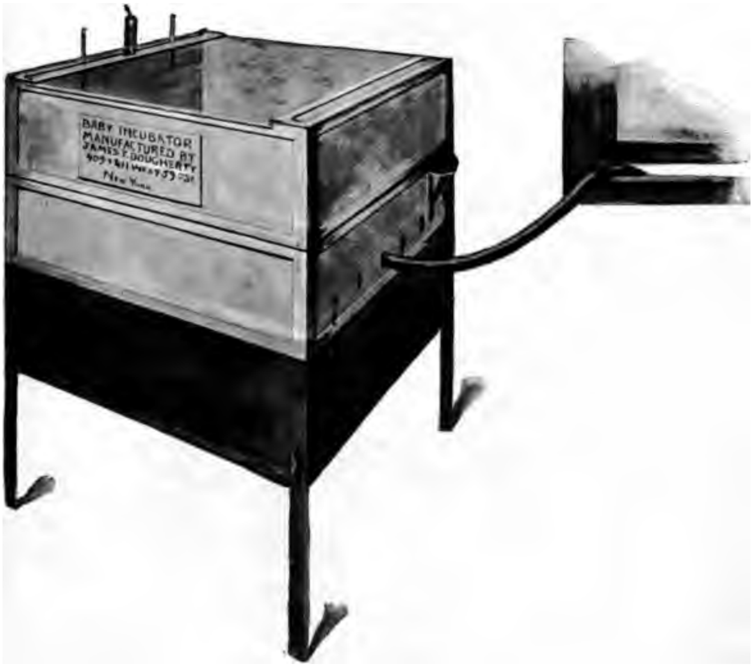
Lion incubator.

We will first take up the methods of maintaining the body-temperature. This is done by means of the incubator.

Incubators.—The simplest model of an efficient incubator for maintaining the temperature of the congenitally weak is that first introduced by Tarnier. Though many complicated pieces of apparatus have been constructed since the time of this clinician, none has surpassed his model in efficiency. The most efficient incubators are made of metal or are porcelain-lined, simple in construction, and allow of thorough ventilation while maintaining the desired degree of temperature. Infections being common at this period, an incubator should be so constructed that it can be easily cleaned and subjected to sterilization before use. Incubators made entirely of wood are therefore useless, if not dangerous.

Of the elaborate incubators that of Lion (Fig. 24) has given the greatest number of successes. This elaborate apparatus can be well ventilated and equably heated. The heat is supplied by radiation. The cheaper forms of incubators are constructed from models of the form which is used in the Sloan Maternity Hospital in New York City (Figs. 25 and 26). In an emergency, any kind of tin-lined

FIG. 25.

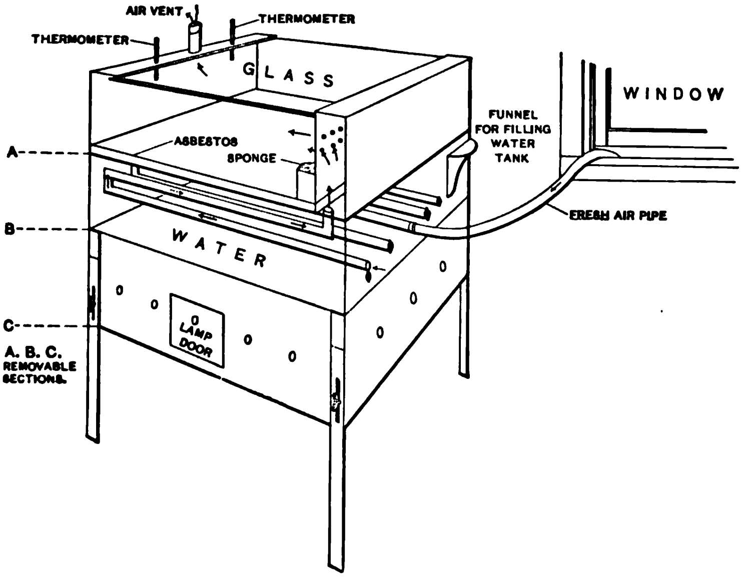


Simple form of baby incubator

box or a basket padded with cotton, supplied with warming bottles, and so protected on top as not to admit of a too rapid escape of the air, answers the purpose of a more elaborate apparatus. In fact, Chapin has shown that with very elaborate apparatus he has had less brilliant results than with simpler means. The cause of his ill-success lies in the fact that complicated apparatus is very difficult to cleanse after having once been infected.

The indications for the employment of any form of incubator are: (*a*) Weight, the infant weighing 2000 grammes or less. Infants weighing 1800 grammes, if vigorous, may be reared without an incubator. (*b*) Subnormal rectal temperature, as has been emphasized elsewhere. (*c*) Cyanosis or sclerema.

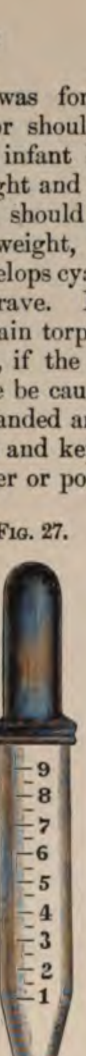
FIG. 26.



Plan of simple form of incubator. A, B, water tank heated by lamp; in this tank are the fresh-air pipes. The air is heated before passing into the top section, in which the infant lives.

The temperature at which the interior of the incubator should be maintained is of the greatest importance. It has been customary to keep the temperature of the interior of the incubator a little higher than that of the infant, with the idea that in this way the heat which is transmitted to the body of the infant is necessary. Later investigations have proved that an infant with a rectal temperature of 30°–32° C. (86.6° to 89.6° F.) will be more comfortable and thrive better in an incubator kept at 25°–26°C. (77° to 78.8° F.) than in one in which the temperature is 35°–37° C. (95° to 98.6° F.), as was formerly practised. Therefore the interior of the incubator should have a temperature of 25° to 26° C. (77° to 79° F.). An infant brought up in an incubator should increase regularly in weight and strength. It should have one or two movements daily, and should take its nourishment at regular intervals. If it loses in weight, remains cold, cannot be roused, breathes superficially, develops cyanosis, dyspnœa, diarrhœa, cough, or vomiting, the outlook is grave. Even should the infant thrive, it must not be allowed to remain torpid. It should be taken out of the incubator cautiously, and, if the respiratory movements are shallow, should from time to time be caused to cry by mild irritation. In this way the lungs are expanded and become aërated. The infant should be turned on its side and kept lying in that position, thus avoiding hypostasis in the lower or posterior part of the lungs. If vomiting occurs, the food should be modified, peptonized, or reduced in quantity, or the intervals of feeding lengthened. Cyanosis, as has been mentioned, is met by friction and flagellation. In carrying this out caution must be observed as regards the liver, which is quite large at this period and easily lacerated. If mucus collects in the throat, it must be cautiously aspirated by means of a small rubber catheter introduced to the back of the pharynx, passing over the epiglottis to the superior opening of the larynx. Success in feeding will also aid in overcoming the cyanosis.

FIG. 27.



Breck's feeding tube for premature infants.

Feeding.—The feeding of premature infants is a most difficult problem. At this time, as a rule, the infant is unable to grasp the breast. Therefore it must be fed with a pipette or a nursing tube constructed for this purpose (Fig. 27). In these cases the milk is pumped from the breast and transferred to the infant. We must be careful not to give too much food, for thereby diarrhœa and vomiting may set in; on the other hand, too little food will only tend to perpetuate the weakness and cause cyanosis. During the first ten days there may be loss of weight, or the weight may remain stationary and finally increase. Budin found in feeding these infants that there were three sets of cases, in each of which he could estimate the amount of food taken daily. In the first set of cases the infants weighed less than 1800 grammes and on the second day took 115 grammes of nourishment; on the tenth day, 320 grammes. The second set of cases were those which ranged from 1800 to 2200 grammes, and on the second day took 128 grammes of breast milk; on the tenth day, 410 grammes. The third set of cases weighed from

2200 to 2500 grammes, and on the second day took 180 grammes of milk; on the tenth day, 425 grammes. Thus, the amount of food will vary with the weight and must be gradually increased in all cases. A small quantity (see Infant-feeding) must be given at each feeding, and the feedings should be at intervals of one and a half hours.

Feeding by gavage instead of by the pipette was first resorted to by Tarnier. It may be said, however, that this is scarcely necessary except in very torpid infants.

We must be exceedingly careful in institutions, in caring for premature infants, to guard against the spread of any form of disease which may attack them. A bronchitis in a premature infant is a more serious disease than in an infant born at full term with normal weight and temperature. This bronchitis is of the infectious type and very fatal to premature congenitally weak infants. As a rule, it leads to bronchopneumonia, and in institutions is apt to spread from one weakling to the other. Any epidemic disease may attack these infants; prophylaxis therefore plays an important rôle in the prognosis. In institutions a congenitally weak infant attacked with bronchitis should immediately be isolated as in any other infectious disease. In private practice visitors should not be allowed to see these congenitally weak infants and thus infect them. Anyone suffering with an ordinary cold should be forbidden to come in the vicinity of an incubator.

Bosi, Giudi, Escherich, and others have proposed the construction of incubator wards, in which the infant should not be exposed to the changes of temperature and danger of infection when taken out of its crib. It may be stated, however, that there is a great disadvantage in the construction of incubator wards, for neither can infections be avoided any more than in ordinary hospital wards, nor can the temperature in a large space be maintained as easily as in small chambers; and, finally, the isolation of one little patient from another cannot be as complete in an incubator ward as in the individual crib or incubator.

Bath and Clothing of the Congenitally Weak.

The congenitally weak or premature infant is easily chilled, and therefore after birth should not be bathed. It should be well anointed with oil, and this removed with absorbent cotton in such a manner that the body is left clean and free from vernix caseosa or extraneous substances. The infant is then wrapped in one layer of sterilized cotton covering the trunk and the extremities. Over this is sewed a jacket of sterile gauze so as to encase the whole body. The buttocks and genitals, however, are left free, so that any meconium or urine that is passed may be caught by cotton placed against these parts. In this way the infant is not chilled when taken from the incubator to be fed or washed.

Ultimate Fate of the Incubator Infant.

Some of the best developed men and women came into the world congenitally weak, so that the physician should spare no effort to bring about success, no matter how weak the infant may appear at the outset. Especially encouraging are the results obtained with the congenitally weak when it has been possible to feed the infant from the beginning to the termination of infancy with breast milk. The statistics of Budin, quoted elsewhere, show conclusively that of the premature infants discharged from his institution with a weight of 2800 to 3000 grammes, those who fared best were the breast-fed infants, of whom only 15 per cent. died before attaining maturity, whereas 41 per cent. of the bottle-fed infants died during infancy.

The physician will have an easier task if, in addition to the incubator, he makes every effort to obtain human milk for the weakling.

Feeding of the Congenitally Weak and Premature Infants.

Breast-feeding.—The ideal method of feeding the congenitally weak, and the one which is attended with the greatest number of successes, is that with breast milk. There are, however, some facts which must not be lost sight of in feeding the congenitally weak on the breast. Their suction power is much below that of the normal infant born at full term. In some cases the congenitally weak infant is unable to nurse at all. If the mother and not a wet-nurse is to nourish the infant, the milk must be pumped from the breast and fed to the child by means of the Breck Feeder, if the infant is unable to nurse the breast directly. In extreme cases the infant will not even have the power to swallow the milk pumped from the breast and fed to it with a feeder. Under such circumstances the milk must be carefully fed to the infant, by means of gavage, four or five times in the twenty-four hours. As a rule, however, the mother of a premature infant will, if the infant is born much before full term, have very little milk in her breast. In such a case, it is advisable to obtain a wet-nurse whose milk is uniform, and whose child is at least one or two months of age. Should a wet-nurse not be available at this time, the infant may be placed on modified milk until the milk appears in the mother's breast. A wet-nurse who nurses a congenitally weak infant exclusively will lose her milk gradually, because the congenitally weak infant, though it nurses the breast, exerts so little suction power that the normal excitation to continued glandular activity of the breast is lacking and the milk gradually diminishes in quantity, finally ceasing to be secreted. It is well, therefore, to allow the wet-nurse to nurse her own infant while supplying the excess of milk to the congenitally weak infant she is caring for. Under this arrangement there need be no fear that either infant will suffer from an insufficiency of milk, inasmuch as the additional stimulus given by the two infants to the gland will result in an increased secretion of milk, a fact which has been repeatedly proved.

The amount of breast milk which a congenitally weak or premature infant will take from the breast will vary widely with the strength, age, and weight of the infant. As a rule, the amount will vary from 200 to 500 c.c. daily. The nursings should be at intervals of an hour to an hour and a half. The younger the infant the more frequent should be nursings and the smaller the quantity at each feeding. If the infant is unable to nurse the breast, the milk may be pumped off and given in a bottle or feeder to the infant; or is given, as has been stated, by gavage. It may happen, as has been intimated, that the mother, after the birth of a premature infant, has very little milk in her breast. If such an infant is placed temporarily on modified milk, the milk may appear in the mother's breast after a week or two, and the gland may be excited to increased secretion by placing the infant at the breast, especially if it be not too premature or weak.

Artificial Feeding.—The feeding of the congenitally weak or premature infant with modified milk is a very difficult task, inasmuch as comparatively few facts are at our disposal to-day as to the success of this mode of feeding. We know that the success attending the feeding of the congenitally weak or premature infant on cows' milk is even less than that of feeding the normal newborn infant. We will illustrate the feeding of these infants by taking as an example a premature or congenitally weak infant born at seven and a half months of pregnancy. Such an infant is first placed upon a mixture containing 1 per cent. of fat, 0.25 per cent. of proteids, and 5 or 6 per cent. of sugar. The infant is given 10 c.c., or 2½ drachms, at each feeding, the intervals between the feedings being one hour. Twelve feedings are given in the twenty-four hours, rest being given for the remaining twelve hours.

After a week of extra-uterine life the percentage of proteids is doubled, the fat and sugar remaining the same. From the fifteenth day of life the infant will be taking ½ ounce at each feeding, twelve feedings being given in the twenty-four hours. After the fifteenth day the proteids may be increased, so that from the thirtieth day of life the infant will be taking a mixture of 1 to 1.5 per cent. of fat, 0.75 per cent. of proteids, and 6 per cent. of sugar, 1½ ounces at each feeding, with intervals of two hours between the feedings. Ten or twelve feedings are given in the twenty-four hours. At this time the infant will have approached the age of a full-term infant. We should now be cautious not to increase the percentages or strength of the mixture too rapidly, but rather to let them remain stationary and watch the increase of weight. If the weight increases along physiological lines, we are then guided by the same considerations which would obtain with an infant born at full term.

Congenitally weak infants, fed upon modified milk mixtures, who show dyspeptic disturbances, evidenced by green stools or white curds in the movements, should a wet-nurse be unavailable, are fed with a peptonized mixture. The peptonizing is carried out with good results by the process detailed elsewhere.

Mixed Feeding.—This is a combination of breast and bottle-feeding in those cases in which the breast does not yield sufficient milk and the weight of the infant remains stationary. This is seen in cases of twins nursed by the mother or even by a wet-nurse. In such cases several feedings by means of the bottle may be given daily in addition to the breast.

The Amount of Food Taken by the Congenitally Weak Infant Daily.—It has been shown conclusively that the congenitally weak infant at the breast will consume daily approximately one-fifth of its own weight of breast milk. The so-called normal quantities of breast milk taken by the congenitally weak infants, carefully weighed before and after nursing, are found by Budin to be as follows:

Infants of	1000	grammes,	200	grammes.
"	1500	"	250	"
"	1800	"	360	"
"	2000	"	400	"
"	2500	"	500	"
"	3000	"	600	"

The amounts of breast milk taken daily in the first ten days of life gradually increase, as stated, from 115 grammes, taken the second day by an infant of 1800 grammes, to 320 grammes on the tenth day. An infant weighing 2200 to 2500 grammes will take on the average 180 grammes, taken the second day, to 425 on the tenth day, its normal quantity of food.

These quantities of breast milk consumed by the congenitally weak will be seen to exceed or equal in amount what the normal infant at full term consumes. This proves distinctly what has always been insisted upon by the writer that the amount of food necessary to the infant is determined by the needs of the body and not by any arbitrary standard of stomach capacity. In other words, the congenitally weak infant, though under weight, really needs more calories of foodstuffs per kilogramme of body-weight than the full-term infant, because it uses up more heat units of energy, having more extent of body surface exposed for its weight than the normal full-term infant. Unless the calories, in the form of increased nourishment, are supplied to these congenitally weak and premature infants, they fail to thrive, become cyanotic, and die. Thus, when feeding these infants with cows' milk, modified or peptonized, it must not be forgotten that the above principles hold true, and that the amount of breast milk consumed by the congenitally weak is a better guide as to the necessary quantity of artificial food to be given these infants than the weight or stomach capacity. On the other hand, if the congenitally weak are fed in excess of their needs, there result vomiting and diarrhœa with loss of weight or stationary weight.

V. ASPHYXIA OF THE NEWBORN INFANT.

Definition and Etiology.—Asphyxia is a condition produced by an interference with the oxygenation of the blood. In the uterus respiration is effected through the placenta. If the placenta is separated wholly or in part from its uterine attachment, or the circulation in this organ is interfered with, the disturbance of the normal conditions causes efforts at respiration, the result of deficient oxygenation of the blood. Asphyxia may thus be produced by tonic contraction of the uterus, premature rupture of the membranes and escape of the liquor amnii, asphyxiation of the mother, a hemorrhage, the administration of drugs to the mother intra-partum, pressure on the cord, injury to the head intra-partum, or through pressure on the vagus intra-partum, with disturbance of the respiratory centres. If the placenta is separated prematurely there are consequent efforts at respiration, during which liquor amnii or mucus may be aspirated and asphyxia thus produced. In the extra-uterine form of asphyxiation the infant is born and makes efforts at respiration; but inherent constitutional weakness, weakness of the respiratory muscles, deformity of the chest, or disease of the lungs renders full expansion of the lungs impossible. Syphilitic disease of the lungs, tumors of the lungs, or affections of the pleura may have the same effect.

Morbid Anatomy.—The blood in infants who have died asphyxiated is thin and fluid. The right heart and large vessels are filled with blood, as are also the sinuses of the dura mater, pia mater, and liver. The liver is dark and bluish in tint. Punctate hemorrhages are found in the pia mater, pleura, pericardium, peritoneum, liver, kidney, retroperitoneal connective tissue, uterus, kidneys, suprarenal capsule, and retina. There is a serosanguinolent effusion into the cavity of the peritoneum, pleura, and pericardium. Œdema of the extremities, scrotum, and connective tissue about the umbilical vessels and pia mater is present. The lungs are dark red and heavy. Ecchymoses are seen underneath the pleura and pericardium. In the lungs there are islands of aërated tissue and areas of atelectasis, even though the infant has breathed. The trachea and bronchi may be filled with liquor amnii, mucus, or meconium ; the latter is recognized by the presence of lanugo, epithelial scales, fatty crystals, bilirubin, and cholesterin crystals. The stomach may be filled with air or meconium.

Symptoms.—If in a normal state when born, the infant breathes energetically, cries lustily, and opens its eyes, and the skin, which is of a purple hue at first, rapidly assumes a pinkish tint. If asphyxia be present, however, we may have two sets of symptoms, which are characteristic of two forms of this condition.

In the first form, or early stage, of asphyxia, the skin has a bluish or pinkish-blue tint. The face is swollen and the conjunctivæ injected. The infant does not move the extremities. The musculature retains its tonicity ; the heart action is slow but forcible ; the apex beat is apparent to the eye ; the vessels of the cord are filled with blood and pulsate ; the respiratory efforts may be shallow and infrequent, or absent ; the infant can be roused and caused to cry.

In the more advanced form of asphyxia the face is pale and waxy, the lips are cyanosed ; the extremities hang lax, and the muscular tonus is absent ; the head falls to one side and the jaw drops. There is no attempt at respiration or only imperfect gasping efforts. The infant has a corpse-like appearance. The heart-beat is weak though palpable. The vessels of the cord are collapsed and pulsation is weak. If a few gasps of respiration are made at birth, these soon cease. On attempt at respiration the ribs are retracted, but the muscles of the face are immobile. Air is prevented from entering the lung by the inspired mucus. The reflexes are absent. If untreated, infants in this stage of asphyxia die. If they live, efforts at respiration must be repeatedly encouraged, else the infants relapse into a torpid condition and the respirations become superficial.

Diagnosis.—Asphyxia must be differentiated from the effects of pressure due to cerebral hemorrhage occurring at birth during a prolonged labor or application of the forceps. In a large hemorrhage death is rapid, but in slight hemorrhage it may be difficult to make a differential diagnosis. If there is a hemorrhage on the surface of the brain, the symptoms may closely resemble those of asphyxia. The breathing is very superficial ; the infant lapses into sopor ; the pulse may at first be slow and subsequently rapid. There may be occasional convulsions. The fontanelle in cases of hemorrhage on the surface of the brain has a peculiar hard feel. The subsequent history only will clear up these cases. Asphyxia may be combined with cerebral hemorrhage. The history of the birth as to the use of forceps and the duration of the labor will aid us. If after irritation the infant relapses into sopor, if the pulse continues slow and there are repeated convulsions, we may assume the existence of hemorrhage.

The **prognosis** in all forms of asphyxia, if untreated, is grave, and in the second stage is necessarily fatal. If treated, however, the majority of these cases recover, especially those in the first stage. As to the cases of the second stage, much will depend on the duration of the second stage of labor and the compression of the cord. The cases in which cerebral hemorrhage of any severity is combined with the asphyxia are grave. Little and Mitchell have demonstrated that idiocy may subsequently develop in these cases.

The **treatment** of asphyxia is directed to clearing the air-passages as much as possible of obstructing mucus, increasing the number of respirations, and stimulating the circulation. The mucus and aspirated meconium are quickly but gently removed from the mouth by the finger.

An instrument has been devised for the aspiration of mucus from the upper part of the larynx and trachea; but this instrument is not always at hand, and a sterilized catheter, No. 7 French, can be easily introduced to the rima glottidis, and the mucus thus aspirated by means of mouth suction. Care, of course, must be taken by the nurse or physician not to infect the catheter. To avoid this a small piece of glass tubing may be attached to the distal end, and in the lumen of the tubing a small piece of cotton may be loosely plugged; thus saliva and bacteria from the mouth will not enter the catheter. Introduction of the catheter into the trachea is hardly necessary.

In order to stimulate the surface, the infant is quickly placed in a bath at 40.5° C. (105° F.), and then in a cold bath, thence transferred to a warm blanket and rubbed thoroughly dry. After this the infant is, if possible, roused by striking the buttocks quite sharply. If these methods do not cause the infant to cry and breathe deeply, artificial respiration by the Schultze method should be resorted to. The operator, standing with his body well balanced, grasps the infant by the shoulders, the thumbs being on the anterior aspect of the thorax, the index fingers in the axillæ, and the other fingers on the back of the chest. The head is supported by the ulner side of the wrists. The operator allows the infant to hang down from his hands between his legs. The infant is then raised or swung upward above the level of the operator's head to the vertical, so that the lower part of the trunk of the infant is bent on the thorax. The thorax is thus compressed, causing passive expiration. The infant is held for an instant in this position, and then swung down to the original hanging position. Passive inspiration is thus performed.

The Schultze manœuvre should be repeated at the rate of about ten times a minute, at intervals of several seconds. Care must be exercised not to injure the thorax by pressure of the thumbs or the other fingers, the infant being swung on the index fingers. After applying the Schultze method as above for a few minutes the infant is given a warm bath, and, if respiration is not completely established, the swingings are repeated. By this method the bronchi and mouth are freed from mucus, meconium, and liquor amnii, if present. The Laborde method is that by which traction is made on the tongue ten on twelve times a minute. The infant is laid on a flat surface with a folded towel placed between the shoulders, and the tongue is rhythmically drawn forward by means of a forceps and allowed to recede a number of times, corresponding to the normal number of respirations.

The mouth-to-mouth method consists in first clearing the upper air-passages of mucus. The operator then forcibly blows into the mouth of the infant. The chest of the infant is then compressed to force out the air (expiration of the infant). This procedure is repeated as often as sixteen times a minute.

The Dew method seeks to accomplish the same result as the Schultze method, but by simpler means. The infant is grasped by the one hand at the nape of the neck, and by the other hand at the knees. The thighs rest in the palm of the hand. The thorax is flexed on the abdomen, and then extension is performed. Alternate expiration and inspiration take place. Inflation of the lungs by means of instruments introduced into the larynx is dangerous. There are other methods of artificial respiration which may be resorted to, such as the Marshall-Hall method, but, on the whole, the Schultze procedure seems the most effective.

The danger in all cases is in abandoning efforts at resuscitation too early. We should persist in our efforts as long as the heart action continues. After the infant has been brought out of the stage of severe asphyxia there is always danger of relapse into a soporous state. In this condition flagellation on the buttocks at regular intervals may be necessary for days.

In some cases, even after resuscitation has taken place, mucus will continue to collect in the upper air-passages. In other words, on account of cardiac weakness there is a persistent pulmonary œdema. In such cases tracheal mucus will collect in the upper part of the glottis, and I have seen brilliant results follow the occasional introduction into the upper part of the glottis of the catheter for the removal of this mucus by means of suction. I have made use of this procedure at very short intervals throughout the twenty-four hours with excellent results.

In cases of asphyxia the after-treatment is as important as the immediate measures. The infant must be constantly watched. If the respirations become too shallow, the infant is gently flagellated; and when mucus collects in the throat, it is removed.

One of the best drugs to help us with these weakly infants is the ammonium carbonate (½ grain) given every two hours, with or without strychnia sulphate ($\frac{1}{500}$th of a grain every three hours). The infant must be kept warm and carefully fed. Some of these infants will not nurse, either on account of inherent weakness or paralysis of the tongue, caused by pressure of the forceps, and much patience must be exercised. If the tongue has been injured or the hypoglossal nerve pressed upon during birth, one side of the tongue may be deflected, and at each feeding the food may find its way into the upper part of the glottis, causing spasms of coughing and cyanosis. In these cases the nurse will discover that the infant can be fed in a certain posture more successfully than in another, or with a pipette instead of the nursing bottle. If the cyanotic attacks are frequent oxygen must be given almost continuously for hours. After being worked over for days, such infants may make a good recovery or die and show extensive atelectasis in spite of the fact that respiration has occurred.

VI. ASPHYXIA SUBSEQUENT TO BIRTH.

In these cases there is no disturbance of the placental circulation previous to the birth of the infant, and therefore no asphyxia. Asphyxia appears after birth as a result of some abnormality in the respiratory apparatus or of disease of the lung, such as syphilitic hepatization ; of pleural exudate ; of compression of the air-passages by a struma ; or of defects of the diaphragm or deficient development of the lungs. In some cases there may have been injury or compression in the vicinity of the respiratory centre.

Prematurity carries with it a pliable condition of the ribs and weakness of the respiratory muscles, an insufficient development of the respiratory centre, and fœtal atelectasis, which give rise to a state of asphyxia. The more premature the infant the more pronounced are these conditions.

Symptoms.—The infant makes no decided effort at respiration after birth. Inspiration is absent or is hardly noticeable and shallow. Râles are absent. The vessels in the umbilical cord are filled with blood and pulsate distinctly. The heart has a normal frequency at first ; then the contractions become slower and may eventually be increased in frequency. The skin is bluish-red in color ; the extremities are cool. If there is any disease or deformity of the lung, the infant dies soon after birth. These cases are only of scientific interest. Of more importance to the physician is the premature infant normal in all respects save in the fact of its expulsion from the uterus before term.

Premature infants at the sixth, seventh, or eighth month are not all born debilitated, nor are all debilitated infants necessarily premature. There are infants born at the eighth month which are as easily reared as an infant at full term. (See The Congenitally Weak.)

VII. ATELECTASIS OF THE LUNGS.

This condition has been referred to in the section on Asphyxia. Atelectasis, or collapse of the lung, may be congenital or acquired. In the congenital variety the infant is either weakly or born prematurely. The respiratory muscles do not possess sufficient tonus to inflate the lung. The result is that the lung remains in the collapsed fœtal state. In the acquired form the lung cannot expand, as a result of obstructions of the bronchi or alveoli, compression of the lung by an exudate in the pleura, deformity of the vertebral column, or aneurysm of the aorta.

Etiology.—The lung at birth is compact, the alveoli being collapsed. The respiratory efforts inflate the alveoli, and the lung unfolds gradually, as described elsewhere. If after birth the respiratory efforts are insufficient and the bronchi obstructed, or parts of the lung compressed or uninflatable, then a greater or less number of the lobuli remain uninflated and atelectasis results.

If part of the lung which is functionating is thrown out of action from any cause, an acquired atelectasis results. This may result either from compression (compression atelectasis) or from obstruction (obstructive atelectasis). A bronchus may be closed or the alveoli may be filled with fluid masses. Atelectasis may result from an accumulation of fluid or air in the pleura, or from an inability of the diaphragm to act in consequence of curvature of the spine, aortic aneurysm, or contracture of the pleura with thickening.

If the whole lung is involved, it is pressed against the spine, condensed and tough, devoid of air, of a pale-red color or pigmented. The areas of partial atelectasis have the same characteristics, but are redder and filled with blood. If a bronchus or bronchiole is obstructed, the lung collapses and returns to the fœtal state. It becomes the seat of passive congestion, so that the atelectatic area is bluish-red in color. Obstructive atelectasis is quite frequent, and is seen accompanying any inflammatory process of the swollen bronchi. The bluish-red atelectatic areas are seen on the surface of the lung to alternate with the red areas containing air. Congenital atelectasis reveals portions of the lung as firm, non-crepitant, dark-blue, depressed areas with a smooth surface on section. These areas can be inflated, and then cannot be distinguished from the surrounding lung. Inflammatory atelectasis shows the same appearances. At autopsies on children dying of inflammatory disease of the lung, these areas of atelectasis are seen more frequently the younger the subject. Rachitic children are especially subject to atelectasis, on account of their inability to inflate the lung completely.

Symptoms.—The symptoms of atelectasis are not always clearly defined. As a rule, the infants, if premature, are weak; their torpid state has been described in the section on the Congenitally Weak.

On the other hand, should atelectasis develop some time after birth as a result of inflammation and plugging of the smaller bronchi, we shall have the combined physical signs of atelectasis, bronchitis, and possibly bronchopneumonia. In this class of cases the physical signs are as follows :

Inspection.—There is intense dyspnœa ; the lower ribs are retracted, and the efforts at inspiration are labored and move the upper part of the thorax less than the lower portion. The surface is pale and sometimes cyanosed. Efforts at coughing are ineffectual, but may bring up a frothy, clear expectoration which adheres to the lips. Sometimes the breathing is quite irregular and catchy, or very shallow ; at times the infant seems to cease breathing.

Palpation is negative except where râles are abundant, when a fine rhonchal fremitus is present. There is little or no vocal fremitus ; it may be increased or it may be diminished, especially in areas designated vesiculo-tympanitic.

Percussion reveals distinct small areas of dulness with a tympanitic note, slight dulness or marked dulness, especially if areas of collapse are present with pneumonia. Sometimes the note over the rest of the thorax, behind especially, is vesiculo-tympanitic. If the areas of collapse are small, no dulness is elicited.

Auscultation.—In areas situated at the apex or toward the base of the lung the air does not seem to enter freely on inspiration, and the expiratory sound is hardly audible (collapse of area) or absent. Breathing is otherwise puerile or exaggerated, rarely bronchial.

Very fine subcrepitant râles are heard in various parts of the lung. Crepitant râles are very distinctly heard in other areas, and are distinguished from the coarser subcrepitant râles by their fine quality. Areas of pneumonia can thus be recognized by the fine crepitations ; the atelectasis, by the absence of respiratory sounds and dulness. Voice sounds vary greatly. When the infant cries the vocal resonance may seem increased, and again normal ; or if the pneumonic area is extensive and is in the vicinity of a large bronchus, we may have tubular resonance.

Temperature is often normal or subnormal ; later, it may be elevated.

Convulsions are common in atelectasis ; in fact, they are peculiar to the disease. They are repeated at frequent intervals, and an infant may have three or four attacks of general convulsions in the course of the twenty-four hours. At the onset of the convulsions the cyanosis increases.

Diagnosis.—The diagnosis is not possible if the area of collapse of the lung be small. If of considerable extent and giving rise to physical signs, the diagnosis may be made.

As a rule, the congenital forms of atelectasis are more extensive than the acquired forms, and thus can be more readily detected.

The diagnosis of post-natal congenital atelectasis will depend upon :

Convulsions.—Given the case of a newborn infant delivered without forceps or force, in the absence of signs of any other disease, such as hemorrhage on the surface of the brain, the presence of repeated convulsions, with cyanosis and dyspnœa in the intervals, the possibility of atelectasis should be considered.

The presence of areas of slight dulness, or tympanitic dulness, or vesiculo-tympanitic resonance all over the chest.

Fine subcrepitant râles.

Still finer crepitant râles.

Areas in which the air enters incompletely.

Prognosis.—There is no reason why an atelectatic area of the congenital variety should not return to the normal if the cause of its existence is removed and the infant regains power to inflate the lung. The same may be said of the acquired form of atelectasis.

Treatment.—The treatment must be directed toward stimulating the heart and increasing the respiratory efforts if the infant is weak or premature. If the heart is weak, the treatment is much the same as in bronchopneumonia. If the infant does not breathe satisfactorily, it is well to make it cry vigorously several times in the twenty-four hours, so that the collapsed area of lung may be inflated and the mucus in the bronchi expelled. Unless made to cry, these infants lie torpid and hardly seem to breathe. The areas of atelectasis are thus increased. If the temperature is subnormal and the infant seems chilled, we may stimulate it by the application of heat externally, either by means of warm baths, hot-water bottles, or an incubator.

VIII. SEPTIC INFECTION OF THE NEWBORN INFANT.

Our views on the subject of septic infection of the newborn have undergone considerable change in the last decade. The former classification of certain processes, such as pyæmia, septicopyæmia, and pyogenic infection, has given way to a greater or less extent to broader views.

By septic infections are meant certain general phenomena produced by bacterial toxins, or by the entry of bacteria into the body by way of the blood or lymphatic channels. The newborn infant is particularly susceptible to infection. At this period of life the ordinary means of defence are lacking, the lymph nodes and spleen are undeveloped, the skin is in a very vulnerable state and is a ready avenue of entrance for bacteria, as are also the mucous membranes. The lack of febrile reaction, also, demonstrates that in the newborn there is little resistance against the invasion of bacteria.

Septic infections may appear under the semblance of a diarrhœa, bronchitis, pneumonia, hemorrhagic conditions, such as Winckel's or Buhl's disease, and dermatitis exfoliativa, all of which are really manifestations of sepsis.

Etiology.—The most frequent causes of septic infection are the pyogenic bacteria, the streptococci and staphylococci. Following these in order of importance are the bacilli of the coli group, the pneumococci, bacilli of general hemorrhagic infection (Babes), the bacillus pyocyaneus (Neumann), the capsule bacillus of Dungern, the bacillus enteritidis (Gärtner), found in hemorrhagic affections resembling Winckel's disease, and the bacillus of Finkelstein, found also in hemorrhagic conditions. The bacteria exist in the air of hospital wards (Emmerich, Babes, Gärtner, Prudden). They are found in the normal breast milk (Neumann), and in the milk of breasts which are the seat of ulceration, fissure, or abscess. The body of the mother, the lochia, and also the liquor amnii after rupture of the membranes, are all sources whence bacteria may gain access to the body of the newborn infant. As a rare source of infection may be mentioned the incubator in which septic cases have been nursed (Allard). The bath-water has been the means of causing epidemics of dermatitis exfoliativa and Winckel's disease among infants in institutions.

Hetero-infection may also be mentioned, such as obtains at the hands of the accoucheur, from unclean instruments and dressings. Among the autoinfections may be mentioned the conditions which obtain in the skin of the infant, which is in process of desquamation. Deprived of its horny layer, which is absent in the newborn, bacteria can penetrate the sudoriparous and sebaceous follicles. Thus, any pustule may give rise to a general or local process.

Umbilical Site.—The umbilical site is not considered as frequent an avenue of infection as in former days when puerperal disease was more common. To-day we have occasional epidemics of umbilical as well as other forms of infection; but with modern methods this form of infection has become more and more infrequent.

Bacteria or their toxins may thus gain access to the body through the intact or wounded skin, the umbilicus, the mucous membranes (buccal or pharyngeal), through the lungs in the respired air, through the digestive tract by means of the food, through the conjunctivæ and the ears, and finally through the genital tract.

Respiratory autoinfections occur through the aspiration of liquor amnii or vaginal secretions. Bacteria may gain access through a minute loss of the lining epithelium of the respiratory tract.

Digestive Infections.—These must be regarded as rare. The manner in which the bacteria gain access to the circulation from the gut has been demonstrated by Booker and Escherich. They have shown that streptococci may gain access to the general circulation by way of lesions of the mucous membrane of the gut. Conjunctival infection, except in specific cases, is rare.

Otogenic Infection.—The ears may be the seat of septic infection, for pus has been found in the ear of the newborn, and thence has entered the general circulation through infection of the lateral sinus, causing sinus thrombosis, meningitis, and encephalitis.

Urogenital Infection.—This may occur by way of the urogenital tract. As first pointed out by Epstein, an inflammation of the vagina, bladder, or kidneys may be a starting-point of general infection.

Among the predisposing causes of infection of the newborn must be considered congenital weakness. Thus, the greatest number of cases occur among the weakly infants of syphilitic or tuberculous parentage, premature infants, and those possessing birth anomalies.

Symptoms.—It is impossible to particularize any form of sepsis so far as the general symptoms are concerned. The reaction in the newborn infant is so imperfect and the signs are so equivocal that it is often only at the autopsy table that the nature of the lesion is determined. It will be convenient, therefore, simply to enumerate the objective changes noted in the various structures of the body in this disease.

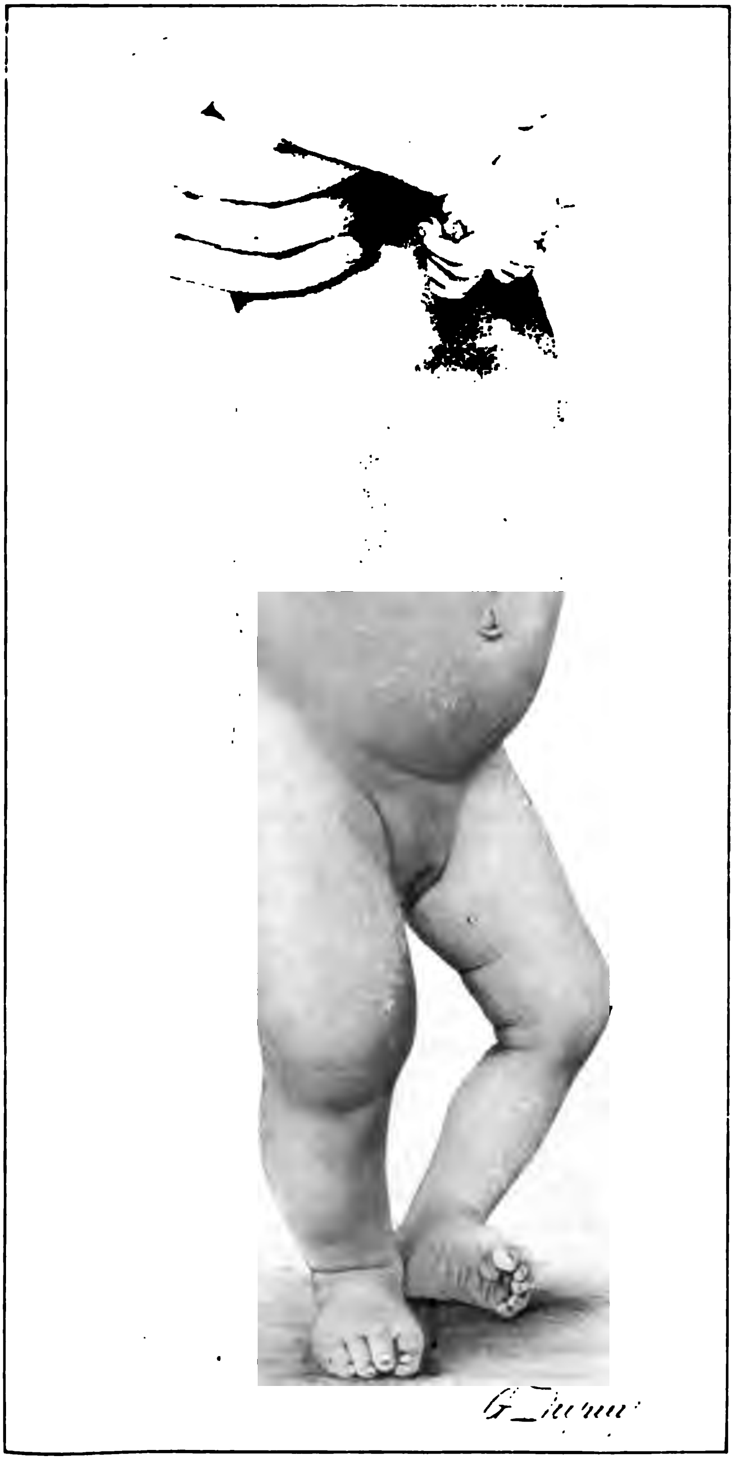
The **skin** may be dry, or the seat of localized œdema or sclerema. It may be the seat of erythema of a polymorphous variety, either on the body or on the extensor surface of the arms or hands. There is sometimes a general or localized cyanosis. A peculiar form of this cyanosis has been described by Finkelstein—the so-called angiospastic cyanosis—in which a central pallor and peripheral lividity are present in the patches. The cyanosis may be limited to the hands and feet.

Eruptions of a pemphigoid character are sometimes seen in cases of sepsis of the newborn infant. The vesicles may be the seat of suppuration, or there may be ulcers and intertrigo varying from superficial erosions to extensive areas of gangrene. The skin may be pale or icteric in hue. There may be erysipelatous patches, furuncles, and abscesses of a multiple variety.

The mucous membrane of the mouth is dry and fissured, and the tongue dry and coated. The roof of the mouth may be the seat of ulcerations superficial or deep, occurring at the median raphe, where we find normally Epstein's pearls, or laterally over the hamular processes of the palate bone (Bednar's aphthæ). The mouth may be the seat of pseudomembranous deposit not due to the diphtheria bacillus (Epstein). In these cases of sepsis sprue may engraft itself on the mucous membrane of the mouth and extend to the pharynx, œsophagus, and stomach. The vagina in female infants may be the seat of catarrhal or pseudomembranous inflammation.

Umbilicus.—Normally, pathogenic bacteria are found about the stump of the desiccating cord, but do no harm; under favorable con-

PLATE V.



Sepsis in the Newborn Infant. Suppuration of the right knee-joint. Osteomyelitis of the epiphyses of the bones forming the joint.

ditions of sepsis, however, these bacteria may increase in numbers and virulence and become a source of great danger. In septic conditions the cord does not fall off promptly. The tissues about the umbilicus are inflamed and the seat of phlegmon and suppuration. Pus may burrow downward toward the bladder along the course of the fœtal structures. The bloodvessels of the cord may be the seat of inflammation, as will be shown later. In some forms of sepsis in which the infectious material may have gained entrance through the umbilicus, the latter may show absolutely no change from the normal.

Bones and Joints.—There may be swelling in the muscles about the joints, as in forms of intramuscular abscess, or the joint itself may be the seat of septic suppuration or so-called osteomyelitis (Plate V.). The shaft of the bone or the epiphysis only may be involved. One or many joints may be the seat of suppuration.

Nervous System.—Functional symptoms, such as apathy, restlessness, or convulsions, may be present, or there may be localized facial paralysis or paralysis of the extremities, traceable to meningitis or encephalitis. Hemorrhages in forms of sepsis may give rise to paresis simulating the traumatic palsies of the newborn.

Respiratory Tract.—The respiratory tract may present catarrhal or pseudomembranous inflammation of the nose, tonsils, larynx, or trachea. The bronchitis and pneumonia, especially in the septic forms of diarrhœa, may be of obscure nature and run an insidious course.

The bronchopneumonia which accompanies sepsis of the newborn is toxic in its nature, with but little febrile reaction and dyspnœa. Pleurisy and abscess of the lung may occur, but are frequently only discovered at the autopsy table.

Circulatory System.—The heart may be the seat of septic endopericarditis. This form of pericarditis is rarely diagnosed.

Stomach and Intestines.—The intestinal tract may be the seat of a septic diarrhœa. There may be vomiting with severe gastro-intestinal symptoms, not infrequently with blood in the vomited matter as a manifestation of toxæmia. In the cases of septic diarrhœa described by Fischl and Czerny there was complicating bronchopneumonia of a severe type.

Liver.—The liver may be the seat of enlargement in cases of extended duration, but the spleen is rarely so.

Urine.—The urine may contain albumin and blood, not infrequently leucocytes and casts, indicating a septic nephritis.

Body-weight.—The body-weight diminishes markedly and rapidly.

Temperature.—The temperature is not characteristic. In the severest forms of sepsis it may be normal or subnormal; in other cases there may be a rise of a degree or more. I have seen this in milder cases. A new complication may be ushered in with a rise of temperature, as often happens with older infants and children, but this is not necessarily so.

Hemorrhages in the Eye.—In some cases examination of the fundus oculi shows the presence of hemorrhages.

Morbid Anatomy.—The changes in the skin have been described. Those of the umbilicus will be found under the section of Umbilical Infection. The appearances in the mouth, nose, and throat have been described, as well as those of the lungs. The alterations in the gastroenteric tract are detailed in the chapter on Diseases of the Gastroenteric Tract.

The liver and kidneys are the seat of parenchymatous or diffuse suppurative changes. The peritoneum is ordinarily intact, although formerly authors believed it to be frequently involved. The pericardium, endocardium, and myocardium may be the seat of slight or marked changes. Microscopical examination of the blood may reveal the infecting bacteria.

Diagnosis.—The origin of some cases of sepsis of the newborn infant is so obscure that not only is a diagnosis made with difficulty, but it is not always possible to determine the point of entrance of the infectious agent. In cryptogenetic cases no lesion may be visible. If an infant cries when it is diapered or washed in the bath, the joints should be examined for suppuration. A pseudomembranous deposit or an ulceration in the mouth is a sign of traumatism with infection. A diarrhœa in the newborn infant is of serious moment. The umbilicus, if swollen or red, should receive due consideration. Lumbar puncture has been proposed for the examination of the cerebrospinal fluid for micro-organisms, but this is hardly justifiable unless a meningitis be present. It has been suggested that the blood should be examined for micro-organisms by means of culture. In several of my cases in which this was attempted it was impossible to obtain the requisite amount of blood sufficient for a culture, the vessels being quite small at this age, and it being hardly justifiable to enter an artery or a very large vein in order to obtain the requisite amount of blood.

Puncture of the spleen for the detection of micro-organisms has been advised. Such a procedure may or may not be advisable, according to the indications in the case.

Course and Prognosis.—Some forms of acute sepsis prove fatal in a few hours. Others, and they are the most common, last from a few days to a week. Others give no symptoms and result in sudden death of the infant. Finally, the subacute cases, which are complicated with progressive emaciation, diarrhœa, and pneumonia, extend over two or more weeks. Septic osteomyelitis and chronic omphalitis are especially protracted. The prognosis in these cases is always grave. Mild forms of intestinal sepsis, after pursuing a short course with fluctuating temperature, may recover completely.

In subacute cases the danger of complications is ever present. Even if bacteria are found to be present in the blood, a recovery is not always impossible.

Treatment.—There is no specific treatment for sepsis in the newborn infant. Prophylaxis is of the utmost importance. The hands of the accoucheur must be as clean in handling the newborn infant as in the treatment of the mother. The cord is tied with precautions described elsewhere. The mouth is not washed. As Epstein has pointed out, Bednar's aphthæ and pseudomembranous inflammations are thus avoided. The nasal passages are not inspected more than is absolutely necessary. The bath-water should be clean and not below 38° C. (100° F.). The food should receive attention. The infant should not nurse a fissured or an inflamed breast. The breast nipple should be cleansed before and after nursing, as stated in the section on Hygiene. The room in which the child sleeps should be ventilated. Contact with the secretions of the mother (lochia) should be avoided.

Therapeutic measures will be directed toward the indication in each case. If a pneumonia or gastroenteritis be present, this complication is treated on the same lines as a primary infection of a similar nature. Osteomyelitis resulting in an accumulation of pus in the joints will receive surgical treatment. Cases complicated by meningitis also will receive the treatment indicated under the section on Meningitis as a Primary Infection. If the indications exist, such as pressure effects, a lumbar puncture may be performed. Abscesses are opened and erysipelatous and purulent skin lesions treated according to surgical procedure in each case.

The strength should be supported, and for this purpose alcohol may be used with small doses of strychnine. The antistreptococcic sera are of doubtful efficacy. The administration of alkalies, such as the salicylate, benzoate, and carbonate of sodium, has been strongly advocated. High saline enemata are advised by Sahli. Subcutaneous and intravenous saline injections have not proved successful.

IX. DISEASES OF THE UMBILICUS.

Diseases of the umbilicus are classified as those which are purely local, such as blenorrhœa, phlegmon, gangrene, and erysipelas; those which begin as a local lesion and result in a general infection, such as arteritis and phlebitis umbilicalis, hemorrhage from the umbilicus; and, finally, those which may be classified as anatomical deficiencies, the herniæ umbilicales.

Omphalitis.

The umbilical cord dries up and drops off in five days, leaving a granulating stump. In the case of weakly infants the cord may not fall off until much later. The stump may become inflamed and pus may form. This, in the majority of cases, is due to infection.

Infection of the site of the ligature of the umbilical cord may easily occur in the newborn, first, because bacteria are normally present, or may be conveyed to the site not only at the time of ligation of the cord, but after the stump has separated and the cord healed. Infection usually takes place at the time of ligation or before the cord separates from the stump. The appearance of the stump in omphalitis varies. In some cases the inflammation is slight, but in others the tissues are red, infiltrated, and coated with necrotic masses resembling pseudomembrane. Numerous small abscesses may be present. The great danger is that the process may involve the umbilical vessels. If the inflammation remains local, recovery is the rule. If the vessels become involved, sepsis may result.

Treatment.—Proper ligation and care in dressing the cord will in most cases prevent subsequent infection. Cleanliness is of the first importance. The hands, instruments, and the tape used for ligation should be scrupulously clean. The care of the stump of the umbilical cord has been a matter of much discussion. The ideal method of dressing the stump has not as yet been found. Some prefer not to bathe the child until after the stump has separated, in order to facilitate the mummification of the cord; others have insisted, if the dressing, hands, and water are clean, that no danger results from the bath, and that a daily dressing of the cord is not improper. This will be taken up elsewhere. If the cord is dressed daily, it should be dusted with some bland powder, such as dermatol, orthoform, or xeroform, even after the stump has separated and until the wound is completely healed, for the site of the umbilical cord is susceptible of infection even after the healing has taken place. The best dressing for the cord is sterilized absorbent gauze several layers thick, and perforated in the centre. The cord is passed through this perforation and enclosed in the gauze. This dressing is renewed daily. If a suppurating surface appears, it should be treated on general surgical principles. As a rule, ointments should be avoided. The ordinary sterilized wet-dressing is sufficient.

Umbilical Fungus.

(*Granuloma.*)

In some cases the stump does not heal after the cord has separated, and a granulating surface which presents a fungoid appearance remains. The granulating mass may become as large as a bean and be pedunculated. There is secretion of pus. The affection is a benign one, and should not be confounded with the so-called *enteratomata*, which are rare. The latter are composed of smooth muscular fibre and tubular glands. These umbilical tumors have been described by Kolaczek, who believes that they are formed by the prolapsus of a persistent omphalomesenteric duct. Von Heukelom asserts that they are intestinal protrusions through true diverticula of Meckel. *Adenoid tumors* of the umbilicus have been described by Lannelongue and Fremont. Hüttenbrenner has reported a *polypoid tumor* of the umbilicus, which he believed to be the remains of the allantois.

Treatment.—If small and flat, the granulations are touched daily with silver nitrate stick and a dry dressing is applied; or the granulations may be carefully scraped off and the stump dressed with sterilized gauze after bleeding has ceased. If the growth is large and pedunculated, it should be ligated at its base with silk or catgut and a sterile gauze dressing applied. In a day or two the mass separates and healing takes place.

Blennorrhœa of the Umbilicus.

Blennorrhœa of the umbilicus is a condition in which there is considerable suppuration and secretion of pus after the stump of the umbilical cord has separated. The area of skin around the umbilicus is red and excoriated. Under proper treatment this condition is curable.

Phlegmon of the Umbilicus.

This is an inflammatory reaction of the umbilical wound due to some local infection. There is an omphalitis umbilicalis. The region of the umbilical wound is red, inflamed, and infiltrated, as is also the neighboring skin. There is pain. The condition may retrograde or the skin may break down and ulcerate, or abscesses may cause infection of the peritoneum. In the latter case the disease is invariably fatal.

Ulcer of the Umbilicus.

The umbilical wound is here replaced by an ulcer of an irregular shape; the neighboring skin is red and swollen, and there is a discharge of discolored pus. There is pain, uneasiness, and fever, or there may be no temperature. In some cases ulcers may exist with a pseudomembranous deposit. The disease, however, has nothing in common with true diphtheria, but is due to wound infection of a streptococcic nature. The umbilical wound may become infected with diphtheria. In such a case the diphtheria bacilli will be found in the discharges and in the membrane of the wound.

Gangrene of the Umbilicus.

This is a very serious condition, and occurs in weak infants amid unhygienic surroundings. The disease may develop in from six to thirty days after birth; the wound becomes bluish or greenish red, discolored, or is converted into a discolored, greenish, bloody mass secreting ichorous pus. The gangrenous process may involve the skin, and usually spreads into the depths of the abdominal wall, involving the urachus, the umbilical vessels, and finally the peritoneum. The prostration is great, and there may be little or no temperature; or the temperature may even be subnormal. Under these conditions there results a general sepsis, and the infant dies of toxæmia or complicating peritonitis. In some cases the gangrenous process begins in the subcutaneous tissues, spreads thence to the peritoneum, the overlying skin remaining tolerably intact. This latter form of necrosis is only discovered and verified postmortem.

Treatment.—The treatment of blennorrhœa of the umbilicus consists in applying some dry dressing with a dusting powder, such as dermatol. By applying this powder daily the condition is generally controlled.

Phlegmon of the umbilicus is treated in the same manner as an ordinary phlegmon, by means of any convenient wet dressing. Liquor Burrowii or Thiersch's solution forms a very convenient dressing.

Ulcer of the umbilicus is treated by means of wet dressing, or by the application of one part of balsam of Peru to four parts of castor oil, applied on gauze. The balsam-and-oil dressing is certainly very agreeable, and successful in many cases.

Gangrene of the umbilicus is treated on surgical principles in the same manner as gangrene in other parts of the body.

Erysipelas of the Umbilicus.

This affection may involve the umbilicus and spread thence to the surrounding skin. It may, however, remain local; but, as a rule, it spreads, involves most of the surface of the abdomen, and in many cases ends in gangrene. If erysipelas remains localized, recovery may result; if it spreads, however, it is generally fatal.

Infection of the Umbilical Vessels.

(*Arteritis Umbilicalis.*)

This is an infection which may take place before or after the separation of the umbilical stump, and may occur by way of the bloodvessels or the perivascular connective tissue of the cord.

In this affection the perivascular connective tissue of the cord may first become infiltrated with serum and be œdematous; later, the various coats of the arteries are affected. Thrombosis results, with disintegration of the thrombi. The lymph-vessels in the connective tissue of the cord carry the infectious material to the various parts of the body.

It must not be forgotten that the normal stump of the cord contains bacteria; to be sure, of the non-virulent type or of reduced virulence. These cause no trouble. Given, however, uncleanliness, either in the dressings or otherwise, these bacteria combined with others may give rise to serious infection. The lochia, though not frequently, may be a source of infection. This infrequency is due to our means of asepsis, and to the protection which our present methods afford against epidemics of umbilical infection.

Umbilical arteritis is a wound infection. It is most frequently seen in institutions, and is the result of implantation of septic matter on the umbilical wound by the hands or instruments, or through the bath-water or unclean dressings. Cases have occurred coincident with the presence of blennorrhœa.

Morbid Anatomy.—There may be simple ulceration with discoloration of the umbilicus and purulent material in the lumen of the artery, with infiltration of perivascular tissue. The vessels running from the umbilicus appear as thickened discolored cords. The perivascular tissue is infiltrated. The process may begin about a centimetre behind the umbilicus and extend downward toward the bladder. The umbilical stump may be normal in appearance or inflamed. The lumen of the arteries contain thrombi. The vessels may be dilated and contain disintegrated purulent masses. There may be lobar or lobular pneumonia, with pleurisy and hemorrhagic infarction of the lung. Parenchymatous inflammation of the liver, kidney, and spleen, and suppuration of one or several joints (see Osteomyelitis) may be observed. Peritonitis may be a complication.

The bacteria found in most of these cases have been streptococci or staphylococci.

There may be metastatic abscesses in the various organs; the tissue of the heart may be the seat of parenchymatous degeneration; the epi- and pericardium may be the seats of ecchymoses and hemorrhages, as also the pleura. There may be suppuration in the cavity of the pleura.

Organs apparently normal in gross appearance may be the seat of parenchymatous degeneration.

Symptoms.—The symptoms of arteritis umbilicalis are often indefinite and give no clue to the cause of the illness. The infants gradually emaciate and succumb, the fatal issue supervening quite suddenly. The umbilicus may in these cases have been long healed and show no evidence of disease; in other cases it is inflamed. There is a sinus leading downward and backward toward the bladder, and from this pus exudes. A tense cord-like structure, the inflamed umbilical vessels, is felt beneath the abdominal wall. Sometimes the first intimation of serious disease is seen in the joints. The mother may tell the physician that the infant cries when it is bathed or dressed. In these cases the knee, ankle, or hip may be swollen, tense, and the seat of exudate. A septic osteomyelitis of the epiphyses of the joint is present, resulting in a suppurative arthritis. As a rule, more than one joint is involved. In other cases the symptoms are indefinite: there is a slight febrile movement. The skin has a slightly gray or icteric hue and may be the seat of erythema or hemorrhages, as mentioned under the heading of Sepsis. There may be a violent gastroenteritis or rapidly fatal pneumonia, or the lung symptoms may be equivocal and not very marked. In other words, there is a sepsis with the symptom-complex of a pneumonia or gastroenteric disturbance.

Hennig's symptom, which consists of a so-called depressed triangle reaching from the umbilicus to the pubis, bounded by red lines indicating the inflamed arteries, accompanied by œdema of the wall of the abdomen, is not always present or to be depended upon.

Course.—The cases may be classed as acute, resembling sepsis and running a very rapidly fatal course, simulating a diarrhœa or pneumonia. Other cases may recover; these are the mild infections. The uncommon cases are those which run a chronic or subacute course with metastatic abscesses throughout the body.

Prognosis.—These cases are generally fatal. A few of the mild cases recover. In these, however, it is a question as to whether the vessels have been involved or whether there was a true infection of a septic nature. The prognosis is especially unfavorable in premature infants.

Phlebitis Umbilicalis.

In this affection the veins which pass from the umbilicus to the liver are the seat of an inflammatory process similar to that affecting the arteries in the affection just described. There is a true phlebitis, with pus in the lumen of the veins, in some cases extending into the liver. The branches of the portal vein are involved. The picture presented is that of metastatic abscesses, as contradistinguished from the parenchymatous degenerations which make up the picture of arteritis umbilicalis. The umbilical vein is dilated and filled with pus; the intima is swollen, inflamed, or eroded; the suppuration extends to the liver, which, with the spleen and kidneys, may be the seat of metastatic abscesses. There is peritonitis of the diffuse variety. Pleuritis, meningitis, and brain abscess may result. There may be abscesses in the skin and also in the joints, the whole picture being that of pyæmia. In some cases the symptoms resemble those of peritonitis complicated with icterus; the respirations are shallow, the abdomen tense, and the thighs are flexed on the abdomen.

Treatment.—It is hardly necessary to say that prophylaxis is in all septic affections the mainstay of the physician. Once inaugurated, infective processes in newborn infants are progressive. In cases of the umbilical type I have advised laying open the structures passing from the umbilicus to the bladder, curetting the sinus thus formed, and inducing healing from the bottom. Recovery has followed in a few exceptional cases. The operation should be performed before general infection has occurred. Van Arsdale operated on one of these cases for me and obtained an apparent recovery—that is to say, the sinus leading from the umbilicus healed and there were no symptoms for weeks after the operation.

In some recorded cases the liver has been incised for abscesses. One case occurred in an infant three months of age, the subject of umbilical phlebitis. The results obtained with antistreptococcic sera have not been encouraging.

Hemorrhage from the Umbilicus.

(*Omphalorrhagia.*)

Hemorrhage from the umbilicus may occur (*a*) from the vessels of the umbilical cord or (*b*) from the umbilical wound itself (parenchymatous).

Hemorrhage from the vessels of the cord may occur if the ligature has not been properly applied; but faulty ligation alone will not account for the hemorrhage in all cases. Runge states that if the cord is cut ten or fifteen minutes after a healthy infant has cried lustily there will be little hemorrhage—certainly not one threatening life. The diminution of arterial pressure in the bloodvessels at this point, due to the establishment of the pulmonic circulation and the natural contractility of the vessels, prevents hemorrhage. The fact that infants among savage peoples and the young of lower animals do not bleed to death, although the cord is not ligated, but simply divided, is thus explained. If an infant, therefore, bleeds after ligation of the cord, the reason must be sought in some physiological or anatomical defect of the bloodvessels. We possess no data which would explain the absence of normal arterial contraction in the vessels of the cord. Inasmuch as this condition may be present during the first days after birth, great care should be taken that the ligature is properly applied. Caution should especially be exercised with premature infants, in whom the bloodvessels are in an embryonal state.

After the separation of the umbilical stump a few drops of blood may be seen on the wound from time to time. This is of no moment. The wound should be dressed with a salicylic powder and amylum (1 : 5), and covered with a dry dressing.

Idiopathic Hemorrhage from the Umbilicus.

(*True Omphalorrhagia.*)

Occurrence.—Winckel, quoted by Runge, has seen only 1 case in 5000 births of true idiopathic hemorrhage from the umbilicus. Males are more frequently attacked than females. I have seen a few cases of this affection.

Etiology.—According to Grandidier, infants *apparently* healthy and strong are for the most part affected. This form of hemorrhage occurs also in infants suffering from congenital syphilis, septic infections, or the acute fatty degeneration of the newborn. In some forms of congenital syphilis there may be hemorrhages into the skin, stomach, intestine, and internal organs. In these cases it is not surprising that hemorrhage should also occur from the umbilicus. Icterus, due to syphilitic affections of the liver and lung, may be present.

In 51 cases of hemorrhage from the umbilicus, Epstein found pronounced septicæmia in 24. The affection is especially prevalent under unhygienic conditions and in foundling asylums. Klebs, Eppinger, Cohnheim, and Weigert have described cases of hemorrhage in which micro-organisms of various kinds were found in the blood and in the hemorrhagic areas. Bacterial colonies were found in the arterial thrombi and in the lungs and kidneys.

The occurrence of hemorrhage from the umbilicus in Buhl's disease is elsewhere described.

Symptoms.—About the fifth day after birth, immediately following separation of the umbilical stump, blood is seen to ooze from the umbilicus. It does not appear to issue from any particular vessel, but oozes from the whole umbilical wound, as from a sponge. The flow may be slight at first and then profuse, or may be profuse from the outset. Pressure upon the wound may cause the hemorrhage to cease, but the flow begins when pressure is withdrawn. In some cases the infants have enjoyed excellent health previous to the hemorrhage. In others there may have been a slight icterus or diarrhœa. However this may be, after bleeding commences cyanosis and icterus of the general surface appear, giving the skin a peculiar bronzed appearance. There are hemorrhages from the stomach and gut. Ecchymoses appear in the vicinity of the umbilicus and on other parts of the trunk. Œdema of the ankle-joints and wrists supervenes. The hemorrhage from the umbilicus is the most characteristic symptom, and cannot be controlled by any means. The blood coagulates very slowly.

Duration.—The disease lasts from a few hours to two weeks. Grandidier's statistics give a mortality of 83 per cent. Death ensues in collapse, with convulsions.

Treatment is directed to controlling the hemorrhage by pressure or by transfixing the umbilical wound. From a study of the pathogeny of this affection, it is evident that no form of local treatment can be successful.

Umbilical Herniæ.

In newly born infants we distinguish two varieties of hernia at the umbilicus.

The first form is of serious character. It is really a hernia of the umbilical cord (hernia funiculi umbilicalis). The condition is due to an arrest of development, as a result of which there is a true defect in the abdominal wall at the situation of the umbilicus. The gut prolapses and is covered by the amnion of the cord and Wharton's jelly, beneath which is the peritoneum. The latter is immediately over the gut. Many of the infants thus affected are premature. In others deformities are present. The hernia is a round or oval tumor of the size of a walnut or an orange, located in the region of the umbilicus, and is continuous with the cord. The sac of the hernia is formed by the peritoneum and amnion. The abdominal walls form the border of the sac. Gut, liver, spleen, kidney, or pancreas may be found in the sac.

If treatment is not instituted at the time of separation of the cord, and the hernia is large, ulceration, gangrene, or septic peritonitis in the sac contents may result.

The second and most common form of hernia in this region is due to a weakness at the point of insertion of the cord. The hernia becomes apparent a few weeks after birth, when the cord has completely cicatrized. It is then noticed that when the infant cries there is a protrusion at this point. The protrusion may be small or large, and is covered by the thin cicatrized skin. The hernia may be central or at one side, or a little above or below the centre of the umbilical ring.

The **treatment** of the first form is purely surgical, and consists in splitting open the sac and sewing the abdominal parietes in apposition. The treatment of the second form is simple. As a prophylactic measure a small pad should be placed on the abdomen, underneath the binder, and should be worn for some time after the stump is healed, in order that there may be no protrusion of the wall and gut during crying spells. If the hernia has taken place, a firm pad, made by enclosing a piece of thick cardboard, one and a half inches in diameter, in a piece of linen, should be applied, and supported by rubber plaster. Another method is to reduce the hernia, fold it inward by means of the apposing abdominal walls, and secure the walls thus brought together with plaster. The plaster should be renewed every three days lest ulceration of the skin result. As soon as the muscles of the abdomen gain strength and the infant is able to stand, the opening at the umbilicus closes and the hernia remains reduced.

X. PERITONITIS OF THE NEWBORN.

Occurrence.—This affection may occur from the first to the seventh day after birth, and sets in, as a rule, with vomiting, pain, as evidenced by crying; diarrhœa, tympanitis, disappearance of the liver dulness, dulness in flanks, showing the presence of fluid in the abdominal cavity. Peritoneal fluid may collect in the pelvis and appear in the scrotum, simulating hydrocele. In such a case the right side of the scrotum is mostly affected, and there is accompanying œdema. The temperature may be as high as 40° C. (104° F.). There are restlessness, emaciation, facies, and death supervenes in from four to five days. Infection is not always limited to the peritoneum: there may be blennorrhœa, phlegmon, erysipelas, hemorrhages, or gangrene of the umbilicus, and with these we may have arteritis, pleurisy, and visceral abscesses. Peritonitis of the newborn may originate at the umbilicus, which is a port of entry for bacteria.

The **prognosis** of these cases is grave; most of them result fatally.

XI. TETANUS OF THE NEWBORN INFANT.

(*Trismus Neonatorum.*)

Tetanus of the newborn is an acute infectious disease or intoxication, strictly speaking, characterized by trismus and tonic muscular spasms, rarely convulsions.

Tetanus of the newborn infant is in the majority of cases due to infection of the umbilical wound by the tetanus bacillus. The bacillus, is conveyed to the wound by means of unclean hands, bandages, or filth of any kind. As a result of the growth of the bacillus ptomaïnes are formed, enter the circulation, and are widely distributed throughout the body. Infection may occur at the time of the ligation of the cord or during the separation of the stump. In 8 per cent. of the cases the disease manifests itself immediately after birth (Hartigan).

Hartigan's assertion that in most cases symptoms appear from the first to the fifth day after the separation of the stump of the cord is incorrect. As a rule, the onset is from the fifth to the twelfth day after birth (Runge). It is rare after the third week. The incubation period in the human subject varies from one to sixty days. In animals which have been the subject of experiment the period of incubation has been but a few hours. Subdural injections in animals have given the shortest incubation period.

Tetanus is common in districts in which uncleanliness in the methods of treating the umbilical cord prevails. It is endemic in the Faroe islands, and is common in the Hebrides, Cuba, and

Jamaica. Negroes, especially, are prone to the malady, on account of their lack of cleanliness in treating the cord. Tetanus of the newborn infant has been demonstrated by Beumer and Peiper to be identical with tetanus in the adult.

Morbid Anatomy.—Beck has described two cases of tetanus with swelling of the motor ganglion-cells, and degeneration of the peripheral portion of the cells with atrophy. There are also changes in the chromatin of the cell. Congestion and hemorrhages in the brain and cord, serous exudates in the cord, and congestion of the internal organs, due to convulsions, are present.

Symptoms.—There is a premonitory period of restlessness. The infants awake abruptly from sleep. They nurse badly, let go of the nipple suddenly, and cry. The peculiarity of the disease in infants is the predominance of trismus, with which the attack begins. The lower jaw becomes rigid and fixed at a distance of a few lines from the upper jaw. It is impossible to introduce the nipple between the teeth. At first there is a tremulous contraction of the muscles of the lower jaw. It is then noticed that the infant is unable to open the jaw, and on slight irritation, either with the fingers or with the breast nipples during nursing, the lips become puckered into the position of playing the flute, and the jaw is contracted and fixed.

The muscles of deglutition become affected, so that swallowing is impossible, and all fluid introduced is returned or rejected. The forehead is wrinkled, and the palpebral fissure diminished. The condition of rigid spasm spreads to the other muscles of the body, such as those of the neck, back, and extremities, and there is opisthotonos. At intervals this spasm relaxes. At the outset, during the intervals between the attacks of rigidity, the body is lax; during such intervals the unfortunates may obtain some rest and take nourishment. These intervals become shorter and shorter, until finally the body is in a state of constant rigidity, resting on the heels and the back of the head. The muscular spasm is a tonic one, called forth by the least irritation, or by sound or a moving body in the room, or even by a draft of air. Dyspnœa with resultant cyanosis is present when the muscles of respiration become affected. Deglutition is impossible. There is no cry, on account of spasm of the laryngeal muscles. The temperature may reach 41° C. (106° F.). In protracted cases it may be normal. The pulse is accelerated. The urine and fæces are passed involuntarily. There is albumin in the urine. The respirations are superficial. The heart action is increased; the pulse may be 200. During a contracture the skin is dark red and cyanotic. Icterus may be present. The face is fixed in expression and œdematous.

Duration.—The disease lasts from a few days to three weeks. Death may ensue in from one to six days from asphyxia or exhaustion. In rare cases the attacks become less and less frequent, and finally cease. Fracture of the bones and rupture of the muscles are among the complications.

Diagnosis.—The diagnosis offers no difficulties. The sudden onset and rigid contraction of the muscles of mastication and deglutition, the intensification of the contractures by the least irritation, the opisthotonos with intervals of relaxation and contraction, the temperature—all tend to aid in the diagnosis. The only question which can arise is that relative to the differentiation of tetanus from contractures with paralysis due to traumatism after birth. In the latter case, however, there will be corresponding pareses, such as are seen in the face.

Again, tetanus may be confounded with cerebrospinal meningitis in the newborn, due to infection with staphylococci, streptococci, or meningococci. In meningitis there is no trismus or tetanic spasms, though there may be rigidity of the muscles of the neck and back. In doubtful cases lumbar puncture will reveal microorganisms of meningitis in the cerebrospinal fluid.

Prognosis.—The prognosis is grave. Baginsky lost all of his cases in newborn infants, while Escherich, Soltman, and Monti report recoveries. Cases which occur late, after separation of the cord, give a better prognosis (Papiewski). Patients die of exhaustion, as a result of sleeplessness, lack of food, and general strain on the nervous system.

Treatment.—Prophylaxis is of the utmost importance in this as in other diseases of the newborn infant. Cleanliness in handling the cord is of the first importance. Escherich cauterizes the stump of the cord, to destroy any bacilli of tetanus which may be present. On the appearance of trismus, the treatment is first directed to the relief of the tonic spasms. Chlorate hydrate in 1-grain (0.06) doses every few hours, by mouth, or by the rectum, is a very useful drug. Calabar bean in the form of the extract is recommended by Monti, who gives $\frac{1}{120}$ grain (0.0005) subcutaneously, repeated until the desired effect is obtained. Cannabis indica, $\frac{1}{2}$ grain (0.03) every two hours, is also given internally. Curare has been used but little with the newborn infant. Of the other remedies, bromide of potassium and trionol have little effect.

Aside from the treatment of tetanus in the newborn by means of drugs, the treatment by means of tetanus antitoxin should be resorted to in every case, in spite of the fact that failures have been recorded by Heubner, Leyden, and Blumenthal. We should inject antitoxin as soon as symptoms appear, inasmuch as favorable cases have been reported by Tizzone, Behring, Engelman, Kohler, and others. The antitoxin is given by means of lumbar puncture. A puncture is made in the ordinary way in the lumbar region, as elsewhere described. Five cubic centimetres of cerebrospinal fluid is allowed to flow off. The syringe is then attached to the puncture-needle and 5 c.c. of antitoxin are injected. Another method is to inject half of the serum by lumbar puncture and the other half subcutaneously.

The use of the tetanus antitoxins has not given satisfactory results in some cases, probably owing to the fact that tetanus is a symptom of advanced toxæmia of the nervous system. In such a condition the action of any antitoxin would be exerted too late to give permament benefit. These patients being unable to swallow must be fed per rectum until the acute symptoms have subsided and deglutition is possible.

XII. ICTERUS IN THE NEWBORN INFANT.

The majority of newborn infants are icteric. Icterus in the otherwise normal newborn infant should be differentiated from that due to sepsis, syphilis of the liver, cirrhosis of the liver, stenosis of the common bile-duct, and yellow atrophy of the liver. Acute yellow atrophy of the liver in the mother during pregnancy may produce an icteric condition in the newborn infant.

Icterus Neonatorum.

An opportunity is rarely afforded to inspect postmortem the viscera of cases of icterus neonatorum, since recovery ensues in the majority of cases. In cases which have come to the autopsy table, all the internal organs, including the bones and cartilages, were icteric. The spleen and kidneys were but little affected, even in severe forms, by the general icteric discoloration. In rare cases the liver was microscopically jaundiced. The intima of the arteries, the fluids in the serous cavities, the pericardial fluid, and the subcutaneous and intermuscular connective tissue have been found to contain bile-pigment and biliary acids (Birch-Hirschfeld). The contents of the gut were normal. The kidneys contained uric acid infarctions.

Etiology.—Icterus neonatorum is as frequent in institutions as in private practice. It is more common among boys (Kehrer). It is seen in premature weak infants, and in those whose birth has been attended by complications. The disease is now traced to both a hæmatogenous and a hepatogenous source. There are certain processes in the blood which also involve the functions of the liver. According to Hofmeier and Silbermann there is a disintegration of red blood-cells in the circulation. These disintegrated red blood-cells are converted by the liver cell into biliary pigment; the solids of the bile are increased, as is also the gross quantity of bile (Minkowski, Naunyn, Stadelmann). It is not known, however, how this increase of bile-pigment gains access to the circulation. One theory (Silbermann) is that with the processes described above certain ferments are set free which cause circulatory disturbances in the liver. Stasis results in the bloodvessels, with consequent pressure on the biliary ducts. Resorption of bile thus follows.

Symptoms.—Fully 80 per cent. of all newborn infants become jaundiced shortly after birth (Runge). The jaundice appears on the second or third day after birth. The icterus may be slight and involve only the face, breast, and back, or may be severe and extend over the whole trunk. In severe forms icterus of the conjunctivæ is present. In this feature icterus neonatorum differs from ordinary catarrhal icterus, in which icterus of the conjunctivæ is the first symptom before the skin is perceptibly tinged. The conjunctivæ are last to be tinged in the jaundice of the newborn. Infants suffering from icterus, though in an apparently normal condition, do not increase in weight as normal infants do, and may even lose ground. When they recover lost weight, they do so slowly.

The urine is brownish at times and contains biliary pigment and acids (Cruse, Hofmeier).

Treatment.—Icterus, neonatorum, if untreated, disappears in three or four days in mild cases; severe cases are more protracted. Neither form needs special treatment.

XIII. THE OCCURRENCE OF HEMORRHAGES IN THE NEWBORN.

Hemorrhages in the newborn are frequent as a result of infection. These hemorrhages may accompany ordinary septic infection and form part of the symptomology of sepsis; or they may assume a characteristic symptom-complex, and, as such, make up a definite picture corresponding to what has been formerly described, and still retained in the text-books for the sake of lucidity, as melæna neonatorum, Winckle's disease, and Buhl's disease. Hemorrhages in the newborn may occur from the nose, the mouth, the conjunctiva, the umbilical wound, the stomach, the intestines, the vagina, the skin, and into most of the internal organs. The causes of such hemorrhages are either congenital hæmophilia, or an underlying dyscrasia, such as syphilis, or septic infection. A congenital hæmophilia is rare and plays but a minor rôle in the causation of hemorrhages in the newborn. Grandidier records only 12 of 575 cases of hemorrhage caused by hæmophilia. In syphilitic infants hemorrhages may occur from two to three days after birth, either underneath the skin, from fissures in the skin, from the stomach, the intestines, or the internal organs. Some contend that in these syphilitic infants, in addition possibly to some infection, there is a change in the arteries; others, deny that such changes exist, and contend that the arterial changes described by Mracek are found in the normal infant (Fischl). I have seen Fischl's preparations and must support his views. Inasmuch as these syphilitic infants come into the world as weaklings, and are on this account susceptible to infection, it is more rational to suppose that if hemorrhages occur they are the result of septic infection. Sepsis, therefore, is the main factor in the causation of all hemorrhages in the newborn. The clinical symptoms of these hemorrhages and accompanying constitutional disturbances will be described under the sections devoted to them. Some forms of hemorrhage have been considered in the sections which treat of sepsis of the newborn, diseases and infection of the umbilical wound, and structures. The remaining forms will now be described, and for the sake of lucidity the early nomenclature is still retained.

XIV. MELÆNA NEONATORUM.

This is a disease of the newborn characterized by a discharge of blood from the rectum and by vomiting of blood. It is a rare affection, occurring about once in 1000 births (Kling, Genrich, Runge). The hemorrhages occur in two distinct conditions:

(*a*) As a symptom of a constitutional dyscrasia. This condition has been treated of under the headings of Hemorrhagic Congenital Syphilis, Sepsis, and the Acute Fatty Degeneration of the Newborn. Runge has shown that not only may the diseases named cause melæna, but that any of the infectious diseases of the newborn may give rise to this condition.

(*b*) The second condition in which melæna occurs is that in which, as Landau, in his monograph on this disease has shown, local lesions, such as erosions and ulcerations resembling ulcus ventriculi, exist in the stomach and gut of the newborn infant. Hecker, Spiegelberg, and others have also described these ulcers of the stomach which produce the symptoms of melæna. Landau attributes the ulcer to embolism resulting from a thrombus of the umbilical vein or the ductus Botalli. Embolism in any artery of the mucous membrane of the stomach gives rise to necrosis and erosion, with the opening up of some arterial branch. Ingenious as this theory is, it is not accepted unreservedly by all, although Landau has proved the presence of emboli in the vicinity of stomach ulcerations. Another theory ascribes the ulcerations to hyperæmia of the mucous membrane in asphyxia and traumatism.

Melæna neonatorum can be caused not only by a coccal sepsis, but by a bacillary infection, as shown by Gärtner, who found a bacillus in the fæces, and in the hemorrhages from the various organs and peritoneum. In other cases it is very probable other microörganisms will be found to have caused the sepsis.

In addition there are cases in which no cause can be found to account for the symptoms.

Morbid Anatomy.—Postmortem examination shows the gastro-enteric tract to be filled with dark hemorrhagic masses. The mucous membrane may be normal, the seat of erosions of greater or lesser extent, or there may be hemorrhagic areas scattered throughout the gut. These may be confined to the stomach or duodenum. There may be true ulcers of the stomach measuring ½ to 2 cm. in diameter, resembling those seen in the adult (Winckel). In some cases the thrombosed or eroded vessel is found in the floor of the ulcer or in its vicinity. All the organs are anæmic, and if syphilis or some other general disease exists there are the changes found in these conditions.

Symptoms.—From two to four days after birth it is noticed that the infant is somnolent or restless; there may be hemorrhagic stools or vomiting of bloody masses, or both these symptoms may be present at the same time. The principal symptom, however, is the bloody stools. These are at first mingled with meconium, and later become frequent and profuse. The vomited matter consists of mucus streaked with blood, or masses of blood of brownish color. The amount of blood lost by the bowel within twenty-four hours may be quite great. Under these conditions death ensues within a period of from twelve to twenty-four hours, with all the symptoms of acute anæmia. In other cases there may be a cessation of the intestinal hemorrhage for from twenty-four to forty-eight hours, but recovery does not always take place, and sudden death from a severe hemorrhage may occur at any time.

The **prognosis** is grave. Sixty per cent. of the infants affected die. The outlook is more serious in conditions of sepsis, syphilis, and acute fatty degeneration than in melæna due to ulcer of the stomach or duodenum.

Diagnosis.—We must differentiate this disease, which is called true melæna, from the so-called spurious form, in which the infant simply passes blood swallowed with the food. This spurious form may occur if the breast nipple is fissured or if there is a fissure of the anus. In other cases blood from the nose or mouth of the infant may be swallowed. Hemorrhages of this kind may occur as part of a general septic infection. In many cases there may be, with other hemorrhages, icterus, cyanosis, œdema, pointing to some general disease. Sensitiveness in the region of the stomach points to ulceration of this organ.

Treatment.—The hemorrhages should be controlled by the application of a cold coil to the epigastrium and the administration of cold drinks. Henoch recommends a drop of liquor ferri sesquichloridi every hour in barley-water. Ergotin is given in doses of ½ to ¾ grain internally or subcutaneously. Suprarenal extract has been administered in some cases which have recovered, and may be tried. In a case coming under my care adrenalin was of no avail. Enemata are not advisable. The heart is stimulated with strychnine, digitalis, camphor, or ether.

XV. ACUTE FATTY DEGENERATION OF THE NEWBORN.

(*Buhl's Disease.*)

This disease, first described in 1861 by Buhl, is an acute parenchymatous fatty degeneration of the liver, kidney, or heart, combined with hemorrhages into the various organs, or from the umbilicus, intestines, or stomach.

Etiology.—The disease occurs in the lower animals, especially in sheep. In the human subject it is a form of septic infection, although in Buhl's cases the vessels of the umbilicus had a normal appearance. Septic infection may occur without any appreciable changes about the umbilicus or elsewhere on the surface of the body (cryptogenetic). The disease is very rare; many cases described as omphalitis and hemorrhage from the umbilicus probably belong to the category of Buhl's disease.

Morbid Anatomy.—The body is icteric or cyanotic; there is œdema of the surface, and not infrequently hemorrhagic areas in the skin. The umbilicus may be covered with blood, but the vessels and wound are otherwise normal. Hemorrhages or petechiæ are found in most of the internal organs, especially the pleura, pericardium, mediastinal tissue, muscles, and mucous membranes. The heart is the seat of fatty degeneration, as is also the liver, which is enlarged. The spleen is enlarged and soft. The kidneys are the seat of fatty parenchymatous changes. The stomach and intestines are filled with blood. There are hemorrhages into the mucous membrane of the stomach and intestine. The intestinal villi are the seat of fatty degeneration.

Symptoms.—The children are born partially asphyxiated. Attempts to resuscitate them are not fully successful. Some die in asphyxia, others after a time have bloody diarrhœal stools. At times there is vomiting of blood, and when the stump of the cord separates there is hemorrhage from the umbilicus. The bleeding from the umbilicus is parenchymatous, and may be so profuse as to cause death. The skin is at first cyanotic, then icteric in hue. Large hemorrhagic areas appear in the skin, conjunctivæ, and mucous membrane of the mouth, and bleeding may occur from the ear and nose. Icterus may become extreme. At times œdema of the surface appears. The temperature is not raised. Death ensues in collapse. The external hemorrhages and icterus are absent in some cases.

Diagnosis.—In the newborn infant this symptom-complex is unique, and must be looked upon as a form of sepsis, either through the umbilicus or through some other avenue. In the newborn infant this disease may be confounded with death from asphyxia. In all cases of medico-legal import the organs should be examined for parenchymatous changes before an opinion is given.

Prognosis.—The disease is fatal.

Treatment.—The physician endeavors to bring the infant out of the state of asphyxia. It can be easily understood that he is helpless in the face of the parenchymatous hemorrhages and degenerations, for which there is at present no remedy.

XVI. WINCKEL'S DISEASE.

(*Epidemic Hæmoglobinuria of the Newborn.*)

This disease, first described in the epidemic form by Winckel, is characterized by the sudden appearance of cyanosis and icterus with hæmoglobinuria.

Etiology.—The etiology of the affection is obscure. Epstein, Strelitz, and Baginsky consider the disease a form of septic infection. Winckel's cases were believed to be due to the use of infected drinking, or bath, water. Birch-Hirschfeld and Strelitz found streptococci in the various organs and the blood. Kamen, in an epidemic of the disease, found the colon bacillus in the capillary bloodvessels and various organs.

Morbid Anatomy.—Postmortem examination reveals no disease of the umbilicus or umbilical vessels. The kidneys are the seat of cortical hemorrhages. The spleen is large and hard, and filled with pigment. There are punctate hemorrhages in almost all the organs, especially in the pleura, pericardium, and endocardium. Hemorrhages are present in the mucous membrane of the stomach and gut, and underneath the liver capsule. Peyer's patches, solitary follicles, and mesenteric glands are enlarged. The liver, heart, and various organs show fatty degeneration. There are bacterial foci in the liver and kidneys. The blood shows an increase in the leucocytes and in the free granules.

Symptoms.—The symptoms in Winckel's cases appear on the fourth day after birth in apparently healthy and well-developed infants. The average duration is thirty-two hours. Some infants succumb in nine hours after the onset of symptoms. Restlessness and cyanosis are first noted. The latter is general, affecting the trunk and extremities. Icterus then develops, and becomes marked within twenty-four hours. The respiration and pulse are accelerated; the temperature may be normal, 38° C. (100.5° F.); the skin is cool. At times there are vomiting and diarrhœa. The urine is passed with tenesmus, brownish in color, and contains blood-cells, hæmoglobin, renal epithelium, granular casts, micrococci, detritus, and ammonium urate. Convulsions close the scene. If the skin is cut, a brownish syrupy fluid exudes.

Diagnosis.—Owing to the similarity of symptoms, Winckel's disease may be confounded with Buhl's disease. The former pursues a very malignant course, and does not present the intestinal and stomach hemorrhages to the same extent as the latter.

Runge and others are inclined to believe that all these hemorrhagic affections are due to a common cause—septic infection. The hæmoglobinuria is simply a marked hemorrhage into the kidney. Parenchymatous fatty degeneration of the various organs is common to both affections.

Prognosis.—The prognosis is fatal.

Treatment.—The treatment is that of sepsis of the newborn.

XVII. SCLEREMA.

(*Sclerema Neonatorum; Scleredema Neonatorum; Sclerema Adiposum.*)

This peculiar and rare affection is apt to be confounded with ordinary œdema. There are two forms of this condition: one form called sclerœdema, or œdematous sclerœdema of Soltman; the second form is called sclerema adiposum, or fat sclerema.

Sclerœdema (Soltman).

This affection is not so rare in institutions on the continent of Europe, although in this country it is uncommon. It is not as common a disease as fat-sclerema, which will be described later. It is a disease of the newborn, and occurs only in the first days of life. Some children, according to Dennis, Billard, and Demme, are born with the disease. In these cases the children are born cold, stiff, cannot move, the surface is swollen, œdematous, tense, a great extent of surface being involved in most cases; and in some cases even an ascites is present. In some, however, the feet are first swollen, then the whole body becomes involved later on. Most of the cases published have been fatal in from a few hours to a few days after birth. The form seen after birth occurs mostly in premature infants, or in the congenitally weak infant, one of twins or triplets, or in infants with a syphilitic history. The disease usually begins four days after birth, or may appear as late as the tenth day or in the third week. These children, as stated, are mostly underweight and congenitally weak.

Etiology.—The etiology of sclerœdema, or acute œdema, is still a matter of speculation. Weakness of the heart, a beginning nephritis, or an infection of some kind, deficiencies in circulation and respiration in premature infants, unhygienic surroundings—all have been advanced to explain this rare condition. In the secondary form, the so-called sclerema adiposum, there is to a certain extent a desiccation of the subcutaneous tissues. Sänger thinks that the excess of palmatin and stearin in the subcutaneous fat of the newborn infant may account for the peculiar solidification, since the temperature is reduced, as it is in fat-sclerema. There are cases of fat-sclerema in which the temperature, as has been stated, is elevated. Such was Barker's case, and I have recently seen such a case, so that the theory of Sänger is scarcely adequate. The cases of fat-sclerema which I have seen have created in my mind the impression of an infectious condition; though this etiology is denied by most investigators. Barker found streptococci in the internal fluids after death.

Symptoms.—There are no prodromata, except possibly an uneasiness on the part of the infant, or dryness and coldness of the surface. The respirations are superficial; the temperature, which falls in most infants after birth, does not return to the normal. When the symptoms are fully developed they are seen first in the lower extremities, in the calves of the legs and the dorsum of the feet, spreading thence to the thighs and involving the suprapubic fat. Rarely the eyelids and both upper extremities are involved. The skin is œdematous, swollen, and much thickened. In some cases the skin does not pit on pressure; in others the pitting takes place, but the skin rapidly returns to the primary condition. The color of the skin is either reddish, if the sclerœdema has appeared before the process of desquamation is complete; whitish, if the desquamation of the skin has been completed; or cyanotic, if the infant is premature. As the disease progresses the skin becomes more œdematous, of a yellowish, transparent color, and in the first form the redness of the skin disappears. In the cyanotic form the cyanosis increases, the skin assumes a bluish, marbled appearance. In the worst forms the skin is so tense that pitting by means of pressure with the fingers does not occur, or immediately disappears when the pressure is released. If the skin is punctured with the needle, there is an escape of fluid or yellowish serum. These infants take the breast badly. They sometimes emit a peculiar, shrill cry, due, it is supposed, to œdema of the vocal cords.

The temperature in the mild forms may range from 34° to 35° C. (93.2°–95° F.); in severer forms, from 30° to 32° C. (86–89.6° F.), but rarely as low as in fat-sclerema, where it may be 22° C. (71.6° F.). A complicating pneumonia, however, may cause a rise of the temperature either to the normal limit or even as high as 41° C. (105.8° F.). The heart is weak; the pulse may have a frequency as low as sixty a minute, and in some cases is not perceptible at the wrist. The respirations are superficial, labored, and slow. The urine contains albumin, sometimes sugar, and, if the infant is icteric, bile pigment; it may also contain red blood-cells, granular casts, and fatty epithelium, and rarely leucin. The disease in most cases is confined to the lower extremities, the mons veneris or suprapubic fat, and buttocks and lower part of the back, but it may spread around to the abdomen, involving its lower part. It seldom occurs in patches or small areas. If improvement occurs, the œdema may disappear, leaving a condition of the skin resembling fat-sclerema. Under these conditions the skin is less wrinkled and œdema of the deeper parts disappears slowly. In fatal cases death supervenes without any marked symptoms. The infants simply fail, the pulse becomes slow, the respiration ceases; children die in apathy and coma.

Duration.—Congenital cases may die in a few hours; the post-natal may linger from four days to two weeks. Complications are rare; they have nothing in common with the primary disease, and result as a consequence of the reduced circulation and liability of these infants to infection. Hemorrhages occur in the lung and pleura; lung complications may occur. Effusions have been found in the peritoneum and pleura; the latter especially in congenital cases. The skin may be the seat of icterus, pustules, ulcers, erysipelas, purpura, or gangrene, especially if complicating sepsis is present. Decubitus ulcers, ecchymoses, and finally pneumonia may occur as a septic complication.

Morbid Anatomy.—So far as the skin is concerned, the œdema post mortem is much the same as during life. The skin, muscles, and cellular tissue, not only of the skin, but of the various regions, such as the mediastinum and vocal cords, are involved. In fatal cases there has been found intestinal catarrh, affections of the lung, such as atelectasis; bronchitis, bronchopneumonia, pleurisy, myocarditis, fatty degeneration of the liver, spleen, and kidneys, hemorrhages into the lung and tissue of the heart.

Prognosis.—Clementowsky, who has made a close study of this disease, has recorded 152 cases with 52 deaths. The presence, therefore, of this disease does not exclude the possibility of a recovery, provided the infant retains a certain amount of constitutional resistance and the disease is not widespread.

Treatment.—The treatment of this form of scleroedema being much the same as that of fat sclerema, both will be treated under a common heading.

Sclerema Adiposum.

(*Fat-sclerema.*)

This condition is much more common than the scleroedema just described, and is not a disease confined entirely to the first days of life, but may occur up to the sixth month of infancy. It is doubtful if the disease occurs as a congenital condition. If so, it is rare. The affection follows or complicates exhausting diseases, and is also seen complicating summer diarrhœa, cholera infantum, and pneumonia. If seen as a complicating condition, it is a forerunner of death. It may be seen not only in bottle-fed, but also in the breast-fed infant, the victim of these affections.

Symptoms.—The disease itself begins mostly in the calves of the legs, but not exactly where the loose connective tissue exists, as the sclerœdema does. The deeper parts are firm; the skin is not movable, and has a doughy feel, as though there were nodules of fat imbedded in the tissues. Another place of predilection of its appearance is in the face, where it is first seen affecting either the tip of the nose or the cheeks. The affection is symmetrical. It appears, as has been stated, in the calves, involves the dorsum of the feet, spreads to the thighs, involves the buttocks, especially the inner parts of the thighs, may spread to the upper extremities, lastly involving the face. The palms of the hands or soles of the feet, even in the severest cases, remain free; as also the scrotum and penis. The skin, when the disease is fully developed, is flat, shining, tense, closely adherent to the subadjacent parts, or it may be of a yellowish, whitish, lardaceous appearance, or may be ecchymotic, cyanosed, or red in areas. When the skin is palpated it has a doughy feel, very much as is seen in a corpse. The skin has lost all its original resiliency. In some cases pitting on pressure may result, but not to the extent seen in sclerœdema. In some cases, where the disease has extended over a large surface, the body may be taken up and will remain stiff and extended like a corpse. The respirations are very shallow and reduced in frequency, 16 to 18 a minute. The heart is weak, its frequency reduced from 80 to 60 or even 30 beats a minute. The temperature is low (much lower than in the sclerœdematous form). It may fall to 30°, 26°, or even 22° C. (86°, 78.8°, or 71.6° F.). If a complicating infection is present, such as pneumonia, the temperature may rise to near the normal. The urine is diminished in quantity, dark, concentrated, contains albumin, casts, urates, and uric acid. If there is a complicating condition, it is generally one of the exhausting diseases, such as summer diarrhœa, cholera infantum, or septic pneumonia. Exitus lethalis, as in the previous form, takes place under conditions of progressive failure of the respiration, reduction of temperature, failure of the heart, unconsciousness, and coma.

Morbid Anatomy.—The skin and subcutaneous tissues post mortem retain the characteristics seen during life. If cut into, no fluid exudes, and very little bathes the surface of the section as compared with what is seen in sclerœdema, where considerable fluid exudes from the cut surface. The tissues are (dry very much like frozen fat). Atelectasis, pneumonia, œdema of the lung, pleuritis, pericarditis, hemorrhages, enlarged spleen, and fatty degeneration of the liver and kidneys may be present as complicating conditions, with or without intestinal catarrh. In the brain, hyperæmia and hemorrhages have been recorded.

Duration.—The duration of the disease is from two to seven days.

Diagnosis.—To diagnose either of these forms from the symptoms just detailed is not difficult; but I have seen it mistaken for the œdema of nephritis. On examination such a mistake can easily be rectified, for in nephritis certain features of sclerema are absent, such as reduction of temperature, lardaceous, corpse-like feel of the skin, the lack of resiliency, especially in the fat sclerematous form. In infants the skin may even retain its original wrinkled appearance, and the deeper tissues of the skin have the characteristics described. On the other hand, nephritis may complicate scleroœdema or fat-sclerema.

Sclerema must not be confounded with a similar disease which occurs in the adult subject and older children. Sclerema of the newborn and scleroœdema do not appear, as in the adult, in patches, but involve whole regions and extremities. This condition of the newborn must not be confounded with sclerodactylia, which is seen in adults and older children.

Prognosis.—The prognosis of fat-sclerema is not necessarily fatal, if primary and not complicated with any exhausting condition; if secondary, as has been stated, it is the forerunner of death.

Treatment.—Inasmuch as these infants have not only a reduced temperature, but a tendency toward a constant progressive reduction of the internal temperature, they should be put in some form of incubator, and the same methods applied as in the care of premature infants. Oxygen is administered to stimulate not only the respirations, but the heart. If the sclerema is not too general the parts may be massaged with camphorated oil; and I have seen some cases in which a decided improvement followed such treatment. Cardiac stimulants are used to arouse the flagging circulation. The best drugs to employ are caffein and strychnin, with or without ammonium carbonate. To these infants we must give very small doses, $\frac{1}{8}$ grain of citrate of caffein every few hours, or $\frac{1}{300}$ grain of strychnin, or $\frac{1}{4}$ grain of ammonium carbonate. In many of these cases the act of nursing is impossible, and they must be fed with the pipette. If unable to swallow they must be fed per rectum. The subcutaneous injection of fluids, in my hands at least, has been of no avail; therefore the hypodermoclysis is of very little utility.

XVIII. OPHTHALMIA NEONATORUM.

(*Conjunctivitis Blennorrhœica.*)

Ophthalmia neonatorum is an inflammation of the conjunctiva, accompanied by a profuse secretion of pus, and in some cases an inflammation of the cornea. It is a specific inflammation of the conjunctiva due to the gonococcus of Neisser. From 30 to 40 per cent. of the children in the institutions for the blind have lost their sight through this disease.

Etiology.—The infant may be infected during labor or after birth. It may be infected immediately after birth, or some time subsequent to delivery. In those cases in which the disease appears from twelve to twenty-four hours after birth, they may be safely said to have been infected in the passage through the parturient canal. In those cases infected after birth the symptoms appear in from three to four days post partum. Finally, children may be infected at any period in the puerperium.

The sources of infection are the secretions from the parturient canal of the mother, or infectious material conveyed to the eyes of the infant by the finger of the nurse or accoucheur. The infection post partum occurs by direct contact of the gonococci with the orbital conjunctiva. In institutions, infections are ten times as frequent as in private practice, where the disease, at least on the continent of Europe, occurs in 0.1 per cent. of births (Silex).

Symptoms.—From three to five days after birth it is noticed that the conjunctivæ are red and swollen; there is an injection of the sclera, swelling of the lids, and increased temperature of the parts, or possibly œdema and profuse secretion, at first of a thin, yellow, serous discharge, which after two days becomes thick and creamy. The swelling of the lids is quite marked; the eyes are closed. In some cases the palpebral conjunctiva protrudes from between the orbital fissure and a profuse creamy pus exudes from between the eyelids. If the child has icterus, this pus may assume an icteric color. The cornea is hazy, covered with secretion, and shreds of pseudomembrane may adhere to the palpebral conjunctiva, especially in the early stages of the disease, thus simulating diphtheritic infection. If not controlled the inflammation of the eye progresses until the whole depth of the cornea is involved, resulting in perforation and prolapsus of the iris, escape of the humor, and consequent panophthalmitis. The constitutional symptoms in these cases consist of a lack of desire to nurse on the part of the infant, and a slightly elevated temperature. If the infant is premature or the subject of any dyscrasia, the constant chilling which takes place as a result of cold applications to the eyes results either in a loss of or stationary weight. Therefore this disease is more serious if it occurs in bottle-fed than in breast-fed infants.

Duration.—The duration of the disease varies according to the intensity of the infection. As a rule, it lasts three or four weeks, when it becomes subacute, and the secretion changes to a mucoid or serous character.

Complications.—The complications, so far as the eye are concerned, are keratitis, with perforation of the cornea and loss of the eye. In some cases arthritis of a gonorrhœal nature has been recorded as a complication; in others, vulvovaginitis may result as a complicating infection.

Diagnosis.—The diagnosis presents no difficulties. There is a simple inflammation of the eyes occurring in newborn infants which is not gonorrhœal in its nature, but in which the local symptoms are not very marked; in fact, so mild as to raise a suspicion at once of its non-specific nature. In other cases of ordinary non-specific conjunctivitis, chemosis, swelling, and œdema of the lids are not marked as compared to what is seen in the gonorrhœal form. The amount of pus secreted is not great, and the course of the disease is, as a rule, benign. We should, however, before deciding as to the innocent nature of a conjunctivitis in the newborn make a spread of the pus on a cover-glass and stain the same for gonococci, as this is the only certain method of determining the nature of the disease. In doubtful cases a culture will be demanded. Clinically, however, the two forms of conjunctivitis are so distinct that we may suspect the one or the other from the mildness or the severity of the local symptoms.

Prognosis.—The prognosis is grave in all cases. A favorable issue will always depend on an early recognition of the disease. If the disease is recognized late in its course, the prognosis becomes not only doubtful, but grave as to the integrity of the organ.

Treatment.—The physician will understand that above all things cleanliness is the first factor in the prevention of this dread affection. In private practice, we will always be able to judge, from a knowledge of the patient and her previous condition, as to the necessity of certain measures, which will be mentioned. If we are cleanly, however, some authors insist that not only in private practice, but in institutions, the severer methods of prophylaxis will remain superfluous. We may state that the principal method of prophylaxis in the past, and at the present day, is the so-called Credé method of prophylaxis of gonorrhœal ophthalmia, and this consists in the instillation of a drop of a 2 per cent. solution of nitrate of silver into the eye immediately after birth. In ordinary cases of head presentation it is contended that if the parts of the mother are cleansed just before the birth of the head, and if after the head is born the eyes are carefully but energetically washed with sterilized water, better results are obtained when large numbers of cases are treated than by the Credé method. Therefore, although in institutions it may be advisable to apply the Credé method, on account of the number of cases which are there treated, it is insisted that in private practice this method remains superfluous. A small dish of sterilized water should be close by, and while one hand supports the crowning head the other should wash the eyes carefully with the sterilized water before the child is born, and the complete washing of the eyes can then be repeated after the birth of the child. By this, the Kaltenbach, method of procedure only 0.3 per cent. of cases to the thousand are infected: whereas the combined results of the Credé method have not been lower than 0.6 per cent. per 1000, on account of the various methods of carrying out the Credé procedure; though Credé himself obtained as low a percentage as 0.1 in 2000 cases.

The disease once inaugurated, the following treatment may be formulated: The eyes are cleansed, every half-hour to an hour, with a 1 : 1000 solution of corrosive sublimate. The eye is opened, and with dry cotton the excess of secretion removed, and then the remaining secretion washed away with the sublimate solution. Small pieces of lint, cut to a size slightly larger than the eye, are kept on ice and applied every two to five minutes. The child is kept warm; otherwise with this treatment the body may become chilled. A solution of 2 per cent. nitrate of silver is dropped into the eye daily. Later, when the secretion of the pus lessens and the conjunctiva is swollen and spongy, a 5 to 10 per cent. silver solution is dropped into the eye and immediately neutralized with salt solution. Any therapy beyond that just outlined is scarcely within the province of the general practitioner; but so important is immediate action in these cases that every practitioner should proceed with the treatment before calling to his aid an ophthalmic surgeon. If one eye alone is affected, it is well to try to save the other eye from infection, and there are several methods by which this may be accomplished. A simple method is to close the eye, cover it with cotton, enough to fill out the hollow of the eye, and then to cover this cotton with a piece of lint. Over this place a piece of gutta-percha protective, and bind the eye shut. Such an eye should be looked at daily before the affected eye is treated and cleansed. Should it become infected, the bandages are removed, and the eye treated in the same manner as the affected eye.

XIX. CAKING OF THE BREASTS.

Caking of the breasts of the newborn is not uncommon, and must not be looked upon as a necessary forerunner of mastitis (Fig. 23).

If the breasts of the newborn are swollen but not very tense they should not be interfered with, as this is caused by an abundant milk secretion, which soon diminishes. No attempt should be made to express the milk. If milk exudes it should be carefully washed off the breast, and the breast protected from traumatism and infection by a pad of dry sterilized gauze. In exceptional cases the breasts seem really tense and painful. Under those conditions they may be gently massaged once a day. The index finger of the right hand is cleansed, anointed with sterilized oil, and the breast is stroked in a circular direction for about five minutes. It is then cleansed and covered with cotton or gauze, as detailed above. It is not possible in the newborn to bandage the breasts tightly, as this procedure interferes with the respiratory movements of the chest.

XX. MASTITIS.

Mastitis in the newborn is the result of infection of the breasts. The organ of one or both sides becomes tense and painful, and the skin covering the breast becomes red or bluish-purple in hue. There are fever and restlessness. After a few days fluctuation appears in the breast, generally toward the base of the nipple.

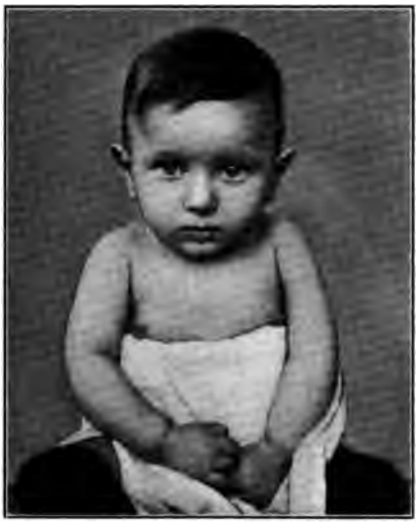
The treatment at first should be directed toward aborting or limiting the inflammation. Nothing is so effective as the application of small squares of lint which have been moistened with a weak solution of sublimate, 1 : 10,000, and applied cold. If after a time fluctuation appears, incision and drainage are indicated.

XXI. INJURIES INFLICTED DURING BIRTH.

Among the injuries incident to birth are those of the face. Pressure of the forceps blade may cause facial paralysis. This, as a rule, disappears in time, though in severe injury of the nerves it may remain permanent. Indentations of the cranial bones may result from the pressure of instruments. In these cases the bone is depressed, and in the space between the scalp and bone there is an effusion. The edge of the bone surrounding the depression is distinctly felt. These depressions need no treatment, as they disappear in time. Traction on the arm may cause a so-called birth palsy, which is the counterpart of Erb's palsy in later life. The paralysis in these cases sometimes remains permanent. Others recover. As a rule, one arm is affected, but in rare cases both arms may be paralyzed. The symptoms are characteristic. In a few days or at a later period after birth it is noticed that the infant does not move one or the other arm (Fig. 28). The affected limb hangs loosely and without power of motion. The fingers or hands may be mobile. The affected arm is cold and the hand may be bluish in tint. After a time atrophy of the muscles about the shoulder-joint may set in. The bony prominences then come into relief. If the arm does not recover power, the muscles continue to atrophy, and there may be subluxation of the head of the humerus at the shoulder-joint. The child in these cases always holds the injured arm with the sound one, in order to protect and support it. At the early period the reactions of degeneration are present, and if the muscles recover, the reaction to the galvanic and faradic current becomes normal. If recovery does not take place, the disappearance of galvanic and faradic irritability of muscle goes hand in hand with the muscular atrophy.

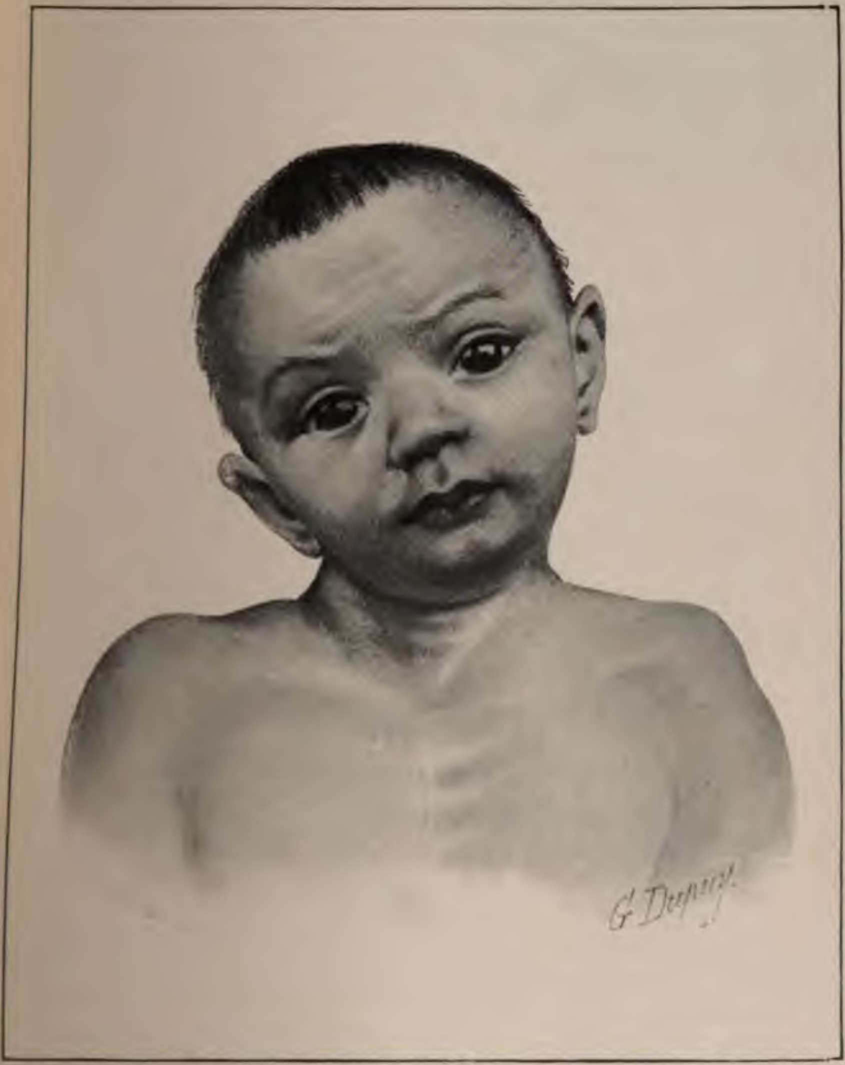
The **treatment** of these obstetrical palsies is similar to that of Erb's palsy. The arm is protected from traumatism. Massage is performed within two weeks after injury, and after four weeks the faradic current is applied to cause muscular contraction. Electricity is applied for a short space of time daily. The progress of these cases can best be judged under treatment. As a rule, recovery takes place in a few weeks. In other cases recovery may be delayed. In a third set of cases recovery never takes place. The galvanic and faradic contractility disappears from the muscle and nerve, and permanent atrophy and disability remain. In these cases there is also retarded growth of the bone. It may be mentioned that in rare cases pressure of the forceps blade has caused a paralysis of the hypoglossal nerve and consequent paralysis of one or other half of the tongue. Every time the infant nurses there will be in such cases great difficulty in swallowing. The infant will cough and become cyanosed. These infants must be nursed slowly or with a pipette until the tongue has recovered power.

FIG. 28.



Birth palsy affecting the left arm, atrophy of the muscles about the shoulder.

PLATE VI.



Hæmatoma of the Sternomastoid Muscle of the Right Side in a Newborn Infant. Swelling at the centre of the anterior border of the muscle; contraction of the muscle with torticollis.

Hæmatoma of the Sternomastoid Muscle.

This affection is the direct result of traumatism during delivery. As a rule, it is seen in cases of breech presentation in which traction has been exerted on the after-coming head. In the majority of the cases coming under my observation the sternomastoid muscle of the right side was affected (Plate VI.). The infant holds the head on one side. The muscle of the affected side is contracted, and the position of the head is that seen in torticollis. A hard nodule is felt along the inner border of the sternomastoid muscle, about the junction of the lower third and upper two-thirds. The tumor is usually the size of a small hazelnut, but may be much larger. Manipulation causes pain. The skin over the tumor is movable and not discolored.

The progress of the affection in all of these cases is much the same. The tumor becomes smaller as the exudate is absorbed, but the torticollis persists, although in time this may disappear. The nature of these tumors is probably that of a hæmatoma caused by rupture of muscular fibres and bloodvessels.

The **treatment** is simple. At first the tumor should be let alone. After a few days gentle massage with the finger moistened with oil is permissible. When the growth hardens the massage may be more vigorous, and be supplemented with an attempt at each sitting to turn the head gently to the opposite side and thus stretch the contracted muscle. Cases which do not recover must be treated by surgical means later in life.

Cephalohæmatoma.

Cephalohæmatoma is an effusion of blood between the pericranium and the skull-cap. The pericranium and scalp are raised into a distinct tumor. In external cephalohæmatoma the effusion is between the pericranium and the skull; in internal cephalohæmatoma it is between the dura mater and the skull. Kee found both forms present in the same patient in 9 out of 20 cases.

Symptoms.—There is a tumor varying in size from that of a hazel-nut to that of an orange, of elastic consistency, situated in most cases on one or the other parietal bone. It is round, elongated, or kidney-shaped. It covers part or the whole of the bone, but never extends beyond the sutures. The skin over the tumor is not sensitive to the touch, is normal or slightly bluish in color, and is perfectly movable over the tumor. After a few days the circumference of the tumor is bounded by a distinct wall, at first soft, but later of bony hardness. The general health of the infant remains good unless there is a complication. This blood tumor appears two or three days after birth. At first it is tense, but afterward becomes softer and doughy to the touch. It reaches its maximum size in from six to eight days. It begins to diminish in the second week, and disappears by the fifteenth week. The tumor is either absorbed or there is a proliferation of bone, which remains as an exostosis. At this time crepitation resembling that of parchment is felt. Around the former tumor a thin wall of bone is found.

Occurrence.—These tumors are not common. Hennig found 230 cases in 53,506 births, or 0.43 per cent. of the whole number. Hofmokl's statistics give a like figure. Most of the cases are vertex presentations. The cephalohæmatoma usually occurs on the right parietal bone, and may follow easy as well as difficult labors. It is present oftener in boys than in girls, and is seen in premature infants as well as full-term babies. It has been observed in breech cases, especially if forceps has been applied to the after-coming head. These tumors may occur on both parietal bones of the infant. In such cases the sagittal suture distinctly separates the two tumors.

Complications.—Internal cephalohæmatoma, or cerebral hemorrhage, may complicate the external tumor. In such cases there has been a difficult labor with the application of forceps. The majority of the infants thus affected die. Suppuration of the tumor may take place, or diffuse cranial phlegmon may result fatally. A section of a cephalohæmatoma shows the scalp to be studded with punctate hemorrhages. The pericranium is bluish and covered with hemorrhages, and is separated from the skull by a collection of fluid blood under great tension. The bone beneath is rough or covered with a few clots. A bony wall is seen around the circumference of the tumor. It is a periosteal formation. After a time the bone and the inner surface of the pericranium become coated with a gelatinous exudate, which is subsequently converted into bone. In some cases quite an extensive bloody effusion is found between the dura and skull.

The situation of the cephalohæmatoma always corresponds to the position of certain natural fissures which exist in the posterior part of both parietal bones, running from the sagittal suture. In the occipital bone these fissures radiate from the lateral fontanelles and separate the upper and the inferior part of the occipital bone.

Pathogenesis.—A cephalohæmatoma is the result of the bursting of a small vessel between the periosteum and bone, and at the situation of the caput succedaneum. Hence the frequent formation of the tumor on the right parietal bone. It is most common in first-born infants. Asphyxia of the infant favors the formation of the tumor. Cephalohæmatoma may also occur as a part of the hemorrhagic symptomatology in general diseases, such as syphilis, sepsis, and Buhl's disease.

The **diagnosis** is made in the presence of an elastic fluctuating tumor distinctly limited by suture and surrounded by a ring or wall.

A caput succedaneum is œdematous and bluish, is seen immediately after birth, passes beyond the sutures, does not fluctuate, and disappears shortly after birth. A hernia of the brain does not fluctuate, grows tense when the infant cries, and shows respiratory fluctuations and pulsation. It can be reduced. Abscess of the scalp is painful, hot, and red; the phlegmon spreads over the whole scalp and is accompanied by œdema of the whole region. If cerebral symptoms are present with a cephalohæmatoma, they point to corresponding internal effusion or cerebral hemorrhage.

The **prognosis** is good if there is no internal tumor or cerebral hemorrhage, or if infection of the external tumor with resulting abscess does not occur. Even the latter, however, does not preclude the possibility of recovery.. The prognosis is bad if the cephalohæmatoma is part of a general hemorrhagic condition, as in syphilis, fatty degeneration, or sepsis.

Treatment.—Uncomplicated cephalohæmatomata are absorbed if let alone. If abscess occurs, the tumor should be opened under antiseptic precautions, evacuated, and the sac packed with iodoform gauze.

On the other hand, even in the early stage, the tumor may be large and tense, and cerebral symptoms may be present. Such effusions of blood may communicate with an internal tumor through the parietal or occipital fissures mentioned. In such very exceptional cases aspiration to relieve internal pressure may be justifiable (Runge).

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SECTION IV.

THE SPECIFIC INFECTIOUS DISEASES.

THE EXANTHEMATA.

THE exanthemata, scarlet fever, measles, Rötheln, varicella, and variola, are acute specific infectious diseases. They form a distinct group. The poison or infectious element originates in the body of the patient. The nature of this poison is unknown. Though suspected to be bacterial, the essential cause in any of the exanthemata has not been isolated. We do know, however, that the acute exanthemata are conveyed from one person to another through the medium of the atmosphere. In this respect they differ essentially from such diseases as typhoid fever, or even syphilis, in which the morbific agent must be introduced into the body. They are therefore contagious in the true sense of the term. Most people are susceptible to some of the exanthemata, such as measles and smallpox. On the other hand, not every one exposed to contagion will contract scarlet fever or varicella. Few persons are attacked twice by the same exanthematic affection, but there are exceptions to this rule. An attack of one disease, such as measles, does not confer immunity from an attack of another, such as scarlet fever. The exanthemata occur either endemically or epidemically. Each has a well-defined period of incubation—that is to say, an interval between the time of the exposure to contagion and the onset of characteristic symptoms. In the different exanthemata this interval varies within wide limits. The period of incubation seems to be more accurately determined in measles than in the other exanthemata. It is well established that two of the exanthemata may occur at the same time in the same subject. This is not a point in favor of the identity of the essential cause of the exanthemata. On the contrary, it is an accepted fact that each of the exanthemata is distinct in itself, and that each disease has its specific essential cause. The exanthemata are characterized by an eruption on the skin, the so-called exanthema, or rash.

I. SCARLET FEVER.

Scarlet fever is an acute infectious disease with a characteristic rash or exanthema. It is highly contagious.

Etiology.—It has not as yet been established whether the infectious agent is a micro-organism, although streptococci have been isolated from the secretions and scales in the desquamative period. Neither do we know whether there is an organism, a protozoan, in the circulating blood. The atmosphere about the patient seems in most cases to be the zone of contagion. The nearer a person has been to the patient the more likely is he to convey the disease to a third person. Articles of clothing may retain the infection for months. Scales from the skin of the patient, dried secretions, the urine if nephritis exists, and fæces are also mediums of infection. The longer the physician remains near the patient the more likely is he to convey the disease. This mode of infection occurs. Osler records his belief in having carried infection to a patient. Foodstuffs handled by those suffering from the disease or by those who have been near patients may convey the disease. This is especially the case with milk, which is said to have been the cause of epidemics in England. The poison of scarlet fever seems to pervade the ward or sick-room for a long time. Whether this period extends over two years, as recorded by Murchison, is a matter not yet settled. We do not yet know how the poison obtains entrance to the body. The discharge from a scarlatinal otitis is said to be capable of communicating the disease.

Susceptibility.—All children exposed to infection do not contract the disease. It is less contagious than measles. On the other hand, although a person may be exposed once and escape, he is not necessarily immune to future exposures. A nurse attended many cases for me before contracting the disease. As a rule, one attack of scarlet fever protects a person from subsequent attacks. The literature records cases of well-observed second and third attacks. The author has met cases of a second attack. We should, however, be cautious in accepting reports of repeated attacks. Rötheln may have been mistaken for scarlet fever.

Occurrence.—Scarlet fever occurs at any age, and in all countries, being endemic in North America and Europe. It is most prevalent in autumn and winter (September to February). It remains endemic wherever introduced. Sporadic cases occur. It occurs also in epidemics. In epidemics only 38 per cent. of the population are affected. There is therefore an immunity of the majority (Jürgensen, on the Faroe Epidemics). As a rule, fully 56 per cent. of those exposed before the twentieth year contract the disease.

Incubation.—According to the German authorities, scarlet fever has an incubation period of from eight to eleven days. English authors (Murchison) fix the period at from three to six days. The vast majority of cases develop within a period of from three to five days after exposure. If eleven days elapse without the appearance of symptoms, we may with reasonable certainty say that the danger is past. Cases of thirty days' incubation are recorded, and the author had a case in his practice in which a physician conveyed the disease, the boy being attacked three weeks after his visit. In all such prolonged periods of incubation, however, there is a probability of a more recent exposure. The contagion is active during the period of incubation and during the eruptive and desquamative stages. The consensus of opinion is that the contagion diminishes in the desquamative stage. We should exercise great caution in allowing convalescents to communicate with the healthy. Strange to say, there are no positive data on this point. Contagion will be treated more fully under Prophylaxis.

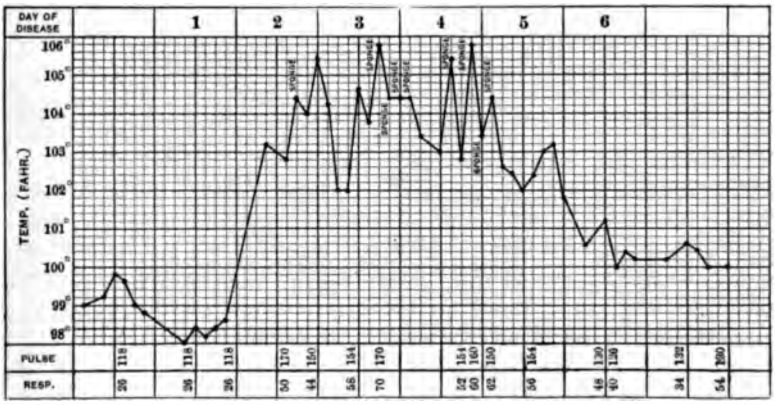
Immunity.—Although there is no absolute immunity at any age, scarlet fever attacks nursing infants less frequently than older children. We have no positive data as to transmission of the affection in utero. Cases are recorded in which the newly born infant has been attacked, but some authors are inclined to look on such cases with doubt. In certain sets of cases the affection takes on a virulent form—cases in families in which all the members attacked will have complications, septic or otherwise, of a fatal character. An instance came under the author's notice in which during a very ordinary epidemic of scarlet fever one family lost two of three children attacked. All had septic malignant fever. There may in such cases be an element of mixed infection (Henoch).

Symptomatology.—Scarlet fever does not present uniform symptoms. A general description of the disease can hardly be given without misleading the student. During an epidemic or during the prevalence of scarlet fever, there are a number of cases of angina in which no exanthema of scarlet fever is seen. This is especially so with those whose duties keep them near scarlet fever patients. There is no doubt that such anginal cases are capable of conveying the disease to others. A case of this kind has come under the author's notice. A nurse suffering from an angina went from a scarlet fever case to a healthy child. Although the nurse had taken all external precautions she conveyed the disease to the child. This raises the question of scarlet fever sine exanthema. Let us say that scarlet fever poison can cause a specific angina capable of conveying the disease to the healthy. Certain forms of exanthema of scarlet fever are very evanescent, and in anginal cases may escape observation.

The *period of incubation* has no fixed symptomatology. In many cases the symptoms begin with the appearance of the eruption. The children play about; they have a slight angina, but do not complain. This is apt to be the case with children who are sufferers from chronic catarrh, enlarged tonsils, or adenoids. In other cases the invasion of the disease is a stormy one. There may be an initial convulsion preceded by a sharp rise in temperature. Examination in such cases may show, previous to the appearance of the eruption, a marked angina or a membranous deposit on the tonsils, but nothing more. Other children suffer from a tonsillitis of moderate severity, a marked febrile movement, and, what is characteristic, attacks of anorexia and vomiting. A chill, followed by fever and vomiting, ushers in a large number of scarlatinal anginas. Occasionally the symptoms of invasion are so mild and evanescent as to escape the notice of even watchful parents. These are the cases in which the first symptom to attract attention belongs to a later period of the disease or to some of the complications. There are thus all degrees in the severity of the symptoms of the period of invasion, varying with the susceptibility of the subject and the virulence of the epidemic.

General Course of the Disease.—An attack of scarlet fever takes a certain general course. After the initial symptoms described, twelve to thirty-six hours elapse, when an eruption or rash appears on the skin: this eruption, though characteristic, varies greatly in intensity, mode of spreading, and distribution. The fever is now very high; the eruption spreads and becomes more intense and general (Fig. 29). At the greatest intensity of the eruption or florescence the fever is highest. In typical cases of scarlet fever the eruption reaches its full development and runs its course within two to six days. At the end of this time it fades, and desquamation begins. The fever subsides gradually, leaving the patient convalescent. The period of invasion is not so sharply defined as in measles, nor is the stage of eruption so distinct and uniform as in that disease. The length of the period of desquamation in both measles and scarlet fever varies.

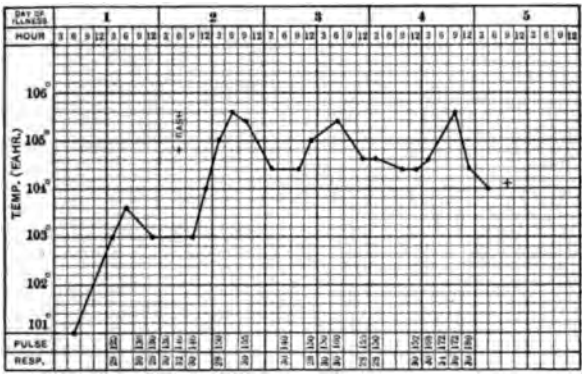
FIG. 29.



Moderately severe scarlet fever; female child four years of age. Normal course. Observed from the outset.

The malignant cases may at first appear mild. The children are taken with vomiting and a moderately high fever, and the eruption appears. While the eruption is spreading, however, the patients become stupid, and within a few hours after the appearance of the exanthema pass into a state of coma. The urine is diminished in quantity or suppressed, and contains blood, albumin, and casts. The temperature remains elevated (Fig. 30). The pulse is rapid and at times thready. These patients remain comatose and die within a few days (three or four) of the onset of the symptoms. In other malignant cases the affection of the throat and adjacent lymph-nodes is a leading factor in the septic phenomena, while the kidneys show very little participation in the general toxæmia. Such patients will show necrotic pseudomembranous inflammation in the fauces after the eruption is fully developed. The glands of the neck are involved. The temperature ranges from 103° to 105° F. (39.4° to 40.5° C.), with daily remissions. The patients have a sallow, septic appearance, and are stupid and irritable. The exanthema fades slightly after having been in efflorescence. The lymph-nodes in the neck enlarge to great size. These patients may die in the second week from general toxæmia. Between the normal course and these malignant forms there are all degrees of severity and mildness in this affection.

Fig. 30.



Malignant scarlet fever; uræmic symptoms from outset. Boy, six years. Sopor increasing to coma; bloody urine. Involuntary passage of urine and fæces. Death in three days after onset of symptoms.

We shall now consider the various phenomena of the disease.

The Angina.—The angina of scarlet fever is limited to the pillars of the fauces, the uvula, the tonsils, and retropharynx. With this there may be a slight suffusion of the eyes. The angina may be simply a slight redness of the fauces and very slight swelling of both tonsils. The lymph-nodes at the angle of the jaw may be very slightly enlarged. The tonsils may be so greatly enlarged as to close the opening of the fauces. This is likely to be the case if there has been antecedent hypertrophy of the tonsils. No membranous deposit may be seen, yet there may be a distinct lacunar form of tonsillitis. The lymph-nodes at the angle of the jaw may be much larger than in the milder anginal cases. The swelling of the lymph-nodes may involve the connective tissue about them in a phlegmonous mass. This is especially so in the severe septic forms of scarlatinal angina of the streptococcus variety.

MEMBRANOUS ANGINA.—Membrane spreading to the pillars of the fauces may be present on one or both tonsils. This condition was formerly called scarlatinal diphtheria. In the vast number of cases of scarlet fever—in fact, in all the uncomplicated cases—this membrane is not a true diphtheria like the diphtheria of Löffler. It is a streptococcus membrane (diphtheroid), caused by the streptococcus of pseudomembranous formations. This membrane may involve the posterior pharynx and nares, and spread downward into the larynx and trachea. True diphtheria of Löffler occurs in those cases of scarlet fever which have been exposed to the infection of diphtheria at or about the time of the outbreak of the scarlet fever or at some period during the course of the disease. The membrane in these cases will show, on examination, the Bacillus diphtheriæ of Löffler. These cases of true diphtheria complicating scarlet fever are exceptional, and presuppose an exposure both to diphtheria and scarlet fever. The pseudodiphtheria is usually caused by a streptococcus of the scarlatinous variety. In some forms of scarlet fever this pseudomembranous inflammation of the tonsils becomes a primary factor in the disease at an early period before the full development of the eruption. This process involves the lymph-nodes and the whole connective tissue of the neck below the jaw in a necrotic streptococcus inflammation. In many cases a true streptococcæmia may result from the entrance of the streptococci into the circulation. In other cases the patient may have passed through the eruptive stage and the process originating in the tonsils may play a leading rôle in the disease. Retropharyngeal abscess, mediastinal burrowing abscess, abscess pointing on the external portion of the neck, or empyema, may result from the necrotic tonsillar affection by extension through the lymph-nodes. Secondarily, a general systemic infection may result in such cases.

The mucous membrane of the mouth presents nothing characteristic in the great majority of cases of scarlet fever. The buccal mucous membrane is pale, and of a normal hue at first; the soft palate may present a few red, irregularly shaped spots or red streaked areas, or these may be absent. Later in the course of the disease a stomatitis may appear. This is more likely to occur in the so-called septic cases. In these the superficial epithelium is removed; the mucous membrane has a dry, red, beefy appearance. The lips are fissured and bleed easily.

The tongue in most cases of scarlet fever is furred at the outset, and may present a slightly reddened appearance at the borders and tip. Only occasionally do we find the so-called characteristic strawberry tongue. This shows an undue prominence and erection of the papillæ of the tongue, especially at the tip. The tip is red, and with the prominent papillæ gives the appearance of a strawberry or of the tongue of the lower animals (cat). In many cases the tongue later becomes denuded of epithelium and shows the erected papillæ on the dorsum; in others it becomes dry and fissured. The latter condition is seen in the fatal toxic cases.

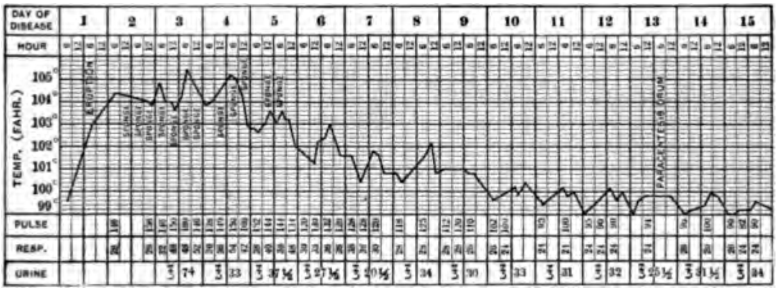
The Exanthema.—The exanthema of scarlet fever, though very characteristic in appearance, varies more than in any of the other exanthemata in mode of appearance, distribution, spreading, and in duration. In the mild cases the eruption is sometimes so evanescent as to escape notice. In other cases it appears only on certain parts of the surface. It may be very discrete in form and punctate. Usually it first appears on the upper part of the chest about the clavicles, spreads down the chest, and around upon the back. At this time it is also seen on the neck, beneath the jaw, behind the ears, and on the temples. It consists of a very finely punctate rose-colored rash. The punctate appearance is the distinguishing feature of the eruption. At the outset this punctate character is best observed on the chest, abdomen, and the nates. If the eruption has in places become confluent, the skin shows a uniform redness. In such cases the punctate character of the rash can best be discovered by studying the skin from a distance in bright daylight. It will then be made out distinctly in those places in which the rash is most recent. A favorite method is to completely undress the patient and study the lower abdomen, the thighs, and nates. In the early cases the punctate character of the rash is apparent on the neck and behind the ears. The appearance of the face at the outset of the disease is characteristic. There is a pallor about the mouth and alæ nasi, while the cheeks are flushed with a flame-like erythema. The cheeks do not show the characteristic punctate rash, although flushed either from the fever or intense dermatitis, which involves the whole surface. The eruption spreads from above downward, involving the arms and forearms, hands, and lower extremities. It retains the punctate character wherever it spreads, but loses this characteristic after it has been out for a short time and become confluent. When confluent the rash causes the skin to appear uniformly red and swollen. The skin is roughened in patches by the erection of the papillæ. In other cases, and especially in those occurring in summer, the skin is studded with myriads of minute vesicles. In other cases the skin may present minute pustules. There is pruritus in the cases in which the dermatitis is severe. The rash of scarlet fever attains its full development at the end of two or three days. It is then said to be in efflorescence. It remains out a variable length of time, in some cases six days. In other cases the eruption may develop fully in two days and then fade. Cases in which the rash is visible for only twenty-four hours are not uncommon. The appearance of a fading scarlet fever rash is very characteristic if it has involved the whole surface. The skin is dotted here and there by raised papillæ, and appears as if irregularly and lightly daubed with rouge. Even a fading rash may be easily diagnosed by an experienced observer. In mild cases the rash may disappear within twelve hours, leaving no vestige of its presence. In other cases the rash appears only on the lower part of the abdomen and upper part of the thighs.

The eruption on the lower part of the extensor surface of the forearms, and also on that of the legs, is apt to assume a blotchy, roseola-like appearance. Such cases have been mistaken for measles.

Abscesses or furuncles, multiple or single, may involve the skin. In rare cases gangrenous processes have been recorded. A secondary infection may be assumed in all of these cases.

The Fever.—In the first few hours there is a rapid rise of the temperature to 104° or 105.8° F. (40° or 41° C.) It remains high with morning remissions until the eruption on the surface reaches its full development. With the fading of the eruption the temperature falls, and within six days, if the case is uncomplicated and typical, becomes subnormal. The patient may show a subnormal temperature for a few days, after which it may rise to the normal. In some cases the temperature may rise very rapidly, reaching its highest point within a few hours. It may then fall to the normal rapidly, though the eruption be still present. Wunderlich and Henoch record cases of profuse exanthema with a mild febrile course or practically afebrile curve, 101.1° F. (38.4° C.), falling rapidly to 100.4° F. (38° C.) within twenty-four hours. In those cases in which there are complications either in the throat, ear, joints (rheumatism), or serous cavities, the temperature-curve will be influenced accordingly. In other cases, evening remissions may occur instead of morning ones. After the fading of the eruption the fever may continue for days, 100.4° to 102.2° F. (38° to 39° C.), in the absence of any complication. After days or weeks of absence of temperature there may occur a distinct rise and a species of relapse similar to that seen in typhoid fever. This is probably due to a form of secondary streptococcus infection. During the height of the eruption the temperature may reach 107° F. (41.6° C.), although in mild cases it may not be over 103° F. (39.4° C.). In cases of septic infection, especially of the lymph-nodes, or in streptococcus diphtheria, with infection of the lymph-nodes, the temperature-curve will be of a remittent character, falling and rising once or twice in twenty-four hours, and may retain this character throughout the affection. Uræmia or any affection of the pleura, lungs, or heart will be ushered in by a rise of temperature even if it has returned to the normal. If a complication occurs early in the disease, the temperature will fail to drop to normal with the fading of the eruption (Fig. 31). In cases of otitis persisting through the stage of desquamation there will sometimes be an evening rise, although the ears are discharging freely. In such cases the bone may be involved (mastoid disease). In severe, malignant forms in which symptoms of profound sepsis, such as coma or stupor, are present from the outset, the temperature remains persistently high (105.6° F., 40.8° C.), remitting a degree toward morning. The temperature remains high until the fatal issue (see Fig. 30).

FIG. 31.



Scarlet fever, moderate severity, in a boy six years of age. Shows the delay in the drop of the temperature due to complicating otitis of the right ear at the outset of the period of desquamation.

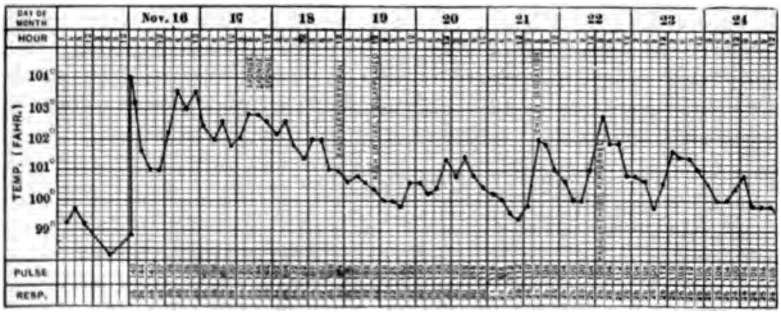
Desquamation.—The period of desquamation begins as soon as the exanthema commences to fade. Generally speaking, since the exanthema first appears on the upper part of the chest and neck we should expect desquamation to begin there. It may be in fine, branny scales, such as are seen in measles; or else, as is most common, the skin peels in larger particles. The hands and feet show the largest scales, and complete casts of the hands and feet are sometimes shed. I have seen the nails shed completely twelve weeks after the attack. The desquamation may be scarcely perceptible. In some cases only certain parts of the extremities, such as the toes or inner portion of the thighs, show desquamation. It is, however, always present. Desquamation in itself is not a pathognomonic symptom of scarlet fever. It occurs in forms of dermatitis which bear no relationship to the disease. It is still a subject of debate whether cases of angina without an exanthema may desquamate. Henoch is inclined to think this possible. We should remember that an evanescent, slightly marked exanthema may escape the notice of even the most careful observer.

The duration of desquamation is variable. I have seen the skin desquamate a second time. The severity of desquamation has no relation to the intensity of the exanthema. Some very marked cases of scarlatina desquamate less than those in which the eruption has been faintly marked.

The Nose.—The close relationship of the nasal passages to the pharynx facilitates the invasion of bacteria from the throat. The nasal passages become affected simultaneously with the severe angina. There is a severe catarrhal or pseudomembranous inflammation of the mucous membrane. In the so-called septic cases there may be an ichorous discharge from the nostrils. There will be in such cases erosions, and sometimes fetor, with the discharge of necrotic tissue through the nasal passages. Necrosis of the cartilaginous and bony structures may result. In other forms there is a pseudomembranous deposit around the opening of the nostrils extending up into the nasal passages. Casts of the nasal passages may be expelled. The membrane may leave a bleeding surface.

Ear.—Duel found the ears affected in 20 per cent. of the cases of scarlet fever. Generally both ears are diseased. Deafness is frequently a result of otitis. Ten per cent. of those who suffer from deaf-mutism can trace their affliction to scarlet fever. Usually the ears become affected in the third week, although they may be involved at the outset of desquamation. The affection of the ears is ushered in by a rise of temperature and manifestations of pain (Fig. 31). Occasionally tinnitus and deafness are initial symptoms. There may be convulsions or even cerebral symptoms. The onset of ear trouble may be insidious, and not suspected until the purulent discharge makes its appearance. If there are premonitory symptoms, they may precede the perforation by one to three days. Ear

FIG. 32.



Female child, two and a half years of age. A mild form of scarlet fever complicated in the second week by an otitis.

complications in scarlet fever are always of serious moment. Meningitis, sinus thrombosis, and abscess of the brain are among the more serious results, and may result long after the fever has run its course. The onset of otitis usually occurs during the period of desquamation. The patient may be up and about. There is still some redness of the throat, with swelling of the lymph-nodes. There is a sudden rise of temperature to 103° or 104° F. (39.4° or 40° C.). The child begins to vomit food and has headache. At night the child starts from sleep and cries as if in pain. Children do not always locate the pain in the ear. The reason is that the pain occurs before the child is quite awake. The sleep is restless. The muscles of the face and hands twitch in sleep. These symptoms may at times abate. The temperature may fall to the normal and then rise sharply. Any of these symptoms should direct attention to the ear.

The *mastoid* may become the seat of inflammation in the fifth or sixth week. The ears may have been discharging very freely. The child is not, however, free from fever. At times during the day the patient complains of frontal headache, is drowsy, and the temperature shows a rise to 102° or 103° F. (38.5° or 39.9° C.). There is tenderness behind the ear or in front of the auditory meatus. There may be a slight blush above and behind the pinna. In these cases the mastoid may be the seat of suppuration. There are forms of otitis which occur on the eighth day of the disease. The temperature does not fall to the normal. The patient has begun to desquamate, but the temperature remains elevated a degree or more and takes fully three or four days longer to fall to 99° F. (37.2° C.) in the rectum than in an uncomplicated case. At the eleventh day of the disease pain is complained of. The drumhead is found to be bulging. An insidious serous otitis media is in progress.

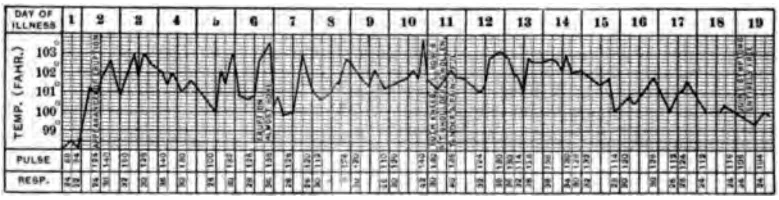
The Eye.—Conjunctivitis may appear in some cases of scarlet fever as a result of a mixed infection. The lachrymal duct is the canal through which such infection travels. Conjunctivitis in cases of gangrenous pharyngitis and rhinitis may lead to panophthalmitis and destruction of the bulb.

Lymph-nodes.—The lymph-nodes in various parts of the body enlarge in scarlet fever. Those situated at the back of the neck behind the posterior border of the sternomastoid muscle may enlarge some days before the appearance of the exanthema. At the time of the eruption we may find the lymph-nodes in the axilla, inguinal region, and those at the angle of the jaw, enlarged. In other cases the lymph-nodes, except those at the angle of the jaw, may not be perceptibly enlarged. In some cases the lymph-nodes at the angle of the jaw may enlarge at the end of the second week, with a distinct rise of temperature to 104° F. (40° C.) or more, as a result of reinfection through the tonsils and pharynx. The connective tissue of the neck beneath the body of the jaw is involved in the inflammation of the nodes. In such cases the swelling has an appearance similar to that seen in angina Ludovici. In severe mixed infection the tissues of the neck may become gangrenous. As a result of such severe gangrenous inflammation, phlebitis erosion into the veins and arteries with fatal hemorrhage may result. Retropharyngeal abscess or retropharyngeal adenitis is a sequence of infection of the lymph-nodes. The retropharyngeal abscess in such cases is not as benign as that occurring independently of scarlet fever. In the latter the abscess is apt to involve a chain of retropharyngeal nodes. Multiple burrowing abscesses result. The nodes of the mediastinum may be affected, causing empyema or pericarditis. The mediastinal abscess may cause death by pressure on the trachea, or, by eroding the trachea, burst into it and cause death through suffocation.

The Mouth.—Stomatitis always occurs in severe scarlet fever. It may be simply a mild catarrhal process. If there is a pseudomembranous formation on the tonsils, this pseudomembrane may spread to the mucous membrane of the soft palate, and the buccal mucous membrane may also become affected. The tongue is dry and fissured; the lips are dry, fissured, and bleed easily. There may be a discharge of necrotic tissue from the mouth. The soft palate, tonsils, and pharynx may be fused into a necrotic mass, emitting an offensive odor.

Joints.—The joints become inflamed in from 2 to 6 per cent. of the cases of scarlet fever. This affection of the joints has been called scarlatinal rheumatism. The joint-affection may, in exceptional cases, precede the exanthema. It appears, as a rule, in the second or third week of the disease (Fig. 33), and is therefore one of the manifestations seen during desquamation. There may be pain in several articulations. In other cases swelling may occur, with effusion of serum into the joints. These cases retrograde. There may be a complicating endocarditis. In other cases there is suppuration of the joint. An arthritis with streptococci in the joint-effusion results. The streptococci invade the joint through the epiphyses of the bone, and produce a streptococcus osteomyelitis with suppuration of the adjacent joints (Lannelongue, Achard, Koplik, Van Arsdale).

FIG. 33.



Boy five years of age, observed from the outset of the disease. Scarlet fever with joint-complications. No cardiac involvement. Recovery.

As a rule, suppuration occurs in only one joint. Cases in which several joints are affected are generally septic, streptococci having gained access to the general circulation through a necrotic focus in the throat or pharynx. Such cases are fatal. There are metastases in the lungs, kidneys, pleura, and pericardium, with hemorrhages in the skin and enlargement of the spleen. Periarticular abscesses rarely occur (Henoch). The prognosis is serious in all suppurative cases.

The Kidneys.—In scarlet fever, as in most infectious diseases, there may be a mild form of nephritis in the earlier stages. There are a small amount of albumin and a few hyaline casts in the urine. This nephritis is of little significance, and has nothing in common with the severer form which occurs later in the disease. The severe form of nephritis begins as a rule in the third week. It has been known to appear in the sixth week. The frequency of this complication varies in different epidemics. In some, only a small number of cases are affected (5 per cent.). In other epidemics fully 70 per cent. of the cases are thus complicated. Its occurrence cannot always be predicted from the severity of the disease. The mildest cases may develop severe nephritis. The diphtheritic forms of angina are more likely to be complicated with or followed by nephritis. On the other hand, the severest forms of scarlet fever may run their course without marked nephritis. Sōrensen has shown that at autopsy the most marked changes may be found in the kidneys, although no clinical signs of the affection have been manifested during life. In 50 per cent. of the autopsies upon scarlet fever patients Friedländer found changes in the kidneys. It was formerly thought that exposure played an etiological rôle in this affection, but this view has been abandoned. Nephritis may develop in cases which have been very carefully guarded from exposure from the outset. Although the symptoms will be detailed elsewhere, it may be here stated that the first symptom is a slight œdema about the eyes and face which spreads to the rest of the body, involving the trunk and extremities, the hands and dorsum of the feet, and the scrotum. In some cases the œdema is not marked, in others the anasarca is extreme. The serous cavities may become the seat of effusion, and there may be hydrothorax, hydropericardium, or ascites. The urine also shows changes very early. The quantity diminishes very rapidly, or it may be completely suppressed. The urine shows the presence of albumin, rarely more than 0.5 per cent. It may be highly colored or smoky, or may be distinctly red in color, owing to the large amount of blood and blood-pigment contained. The urine in cases of partial or complete suppression generally contains a large amount of albumin, blood, hyaline, epithelium, and blood-casts, renal epithelium, and leucocytes. The specific gravity may at first be high, 1.030; later, when diuresis is inaugurated, it may fall to 1.006. All cases do not run their course with anasarca.

Henoch has seen cases without this symptom. The invasion of the affection is sometimes marked either by a rise of temperature or convulsions. The prognosis is good in spite of the very alarming symptoms, such as convulsions and coma, which are seen in some cases. This nephritis usually runs its course in from four to six weeks, leaving the kidneys intact. Sometimes the nephritis apparently subsides, but albuminuria of a very mild or intermittent form persists for months. In fact, many of the so-called cases of paroxysmal albuminuria are probably due to unnoticed scarlatinal nephritis. Finally, the author has seen cases in which the anasarca recurred at long intervals as a result of chronic diffuse nephritis.

Uræmia.—Uræmia commonly sets in with a diminution in the whole quantity of urine passed daily. It may supervene without any distinct change in the quantity or quality of the urinary excretion (Henoch). In these cases the changes in the urine follow the appearance of the uræmic symptoms. Uræmia may also appear notwithstanding the passage of an increased amount of urine. The latter mode of onset in uræmia is very uncommon. The early symptoms are vomiting, headache, and slight twitching of the facial muscles. These may subside with the abatement of the nephritis. We may have, however, eclampsia as the first symptom, with tonic or clonic convulsions, unconsciousness, and coma with temporary absence of the reflexes. The respirations are increased, and in most cases the temperature rises. The pulse is small and the skin dry. The convulsions may subside, but the coma may continue. The eclamptic seizures may be repeated. The uræmia may subside, and after a very protracted interval reappear with a repetition of the above phenomena. Mania, melancholia, and aphasia may ensue.

Amaurosis without changes in the retina is a more common condition. The retinitis of Bright's disease is absent in scarlet fever. Litten found a swollen condition of the papilla. Amaurosis may persist in the intervals between the convulsions.

The heart action immediately preceding the convulsions is slow. The pulse may be as low as 40 per minute. During the convulsions the heart action is increased. The respirations may be 60 and the pulse 200 (Jürgensen).

The temperature may be 100.4°–103° F. (38°–39.5° C.), rarely 107.6° F. (42° C.), with an initial chill (Jürgensen).

Uræmia may set in at any time while the kidney is affected.

The Heart.—Myocarditis of an acute infectious character is likely to supervene in septic cases of scarlet fever. The changes in the myocardium may also be secondary to changes in the pericardium and endocardium.

Endocarditis of the cardiac walls is more frequent than that of the valves. For this reason murmurs should be carefully observed.

No conclusions as to their valvular origin can be reached until long after convalescence. Endocarditis is uncommon, but is more frequent in this disease than in diphtheria or typhoid fever.

Pericarditis is rare. Muscle murmur is often mistaken for it. If present, pericarditis is usually of the dry fibrinous or serofibrinous variety. It is rarely purulent, except in cases of marked purulent involvement of other organs and cavities, notably the pleura.

Dilatation of an acute character may supervene early in severe cases. In such cases we may have tachycardia or bradycardia. There may be cyanosis. Sudden death is very rare in scarlet fever.

Friedländer has shown that in scarlet fever with marked nephritis and uræmia, the consequent increased arterial tension results in dilatation of the left ventricle, with slight hypertrophy. The weight of the heart is increased 40 per cent. The pulse may be slow and irregular. As the nephritis subsides the tension diminishes and the frequency of the pulse increases. Hypertrophy being the result of long-continued increased tension, can be demonstrated only in extreme cases. Dilatation is rarely so great as to cause death.

Lungs.—The lungs may be affected by pneumonia, which is generally of the bronchopneumonic type. Lobar pneumonia as a complication of scarlet fever is rare. Gangrene of the lung may occur in the severe septic cases.

Pleura.—Pleuritis as a complication of scarlet fever usually appears in the middle of the second week. It is commonly of the serous variety, but the author has had many cases in which there was an empyema usually of the streptococcic variety. Fürbringer has shown that in 5 per cent. of the cases of pleurisy there is nephritis.

The Blood.—There is a diminution of the hæmoglobin, which is marked in cases in which nephritis is present. During convalescence the hæmoglobin increases. Slight leukocytosis is also present in the course of the disease. There may be purpura and surface hemorrhages.

Stomach and Intestine.—Vomiting has been mentioned as an early symptom in scarlet fever. It is sometimes repeated in the course of the disease if a cough due to any laryngeal or pulmonary complication exists. Diarrhœa is sometimes a serious complication. There may be a simple diarrhœa, in which an excessive number of movements may threaten the life of the patient early in the disease; or, on the other hand, the diarrhœa may subside without serious results. The diarrhœa may take on a dysenteric or typhoidal type, with severe hemorrhages from the gut. There are some forms of diphtheria of the pharynx, stomach, and large intestine in the septic types of scarlet fever which have been described by Litten.

Sequelæ.—As sequelæ to scarlet fever may be mentioned:

Anæmia, which may persist for some time.

Glandular Swellings.—The lymph-nodes at the angle of the jaw are apt to remain enlarged long after convalescence. The tonsils may remain large.

Tuberculosis may follow scarlet fever. It cannot be said that there is any distinct connection between the two diseases. Scarlet fever may leave the patient more susceptible to infection either of acute miliary or chronic tuberculosis.

Nervous Diseases.—Chorea has been noted by Gerhardt to follow scarlet fever, as have also rheumatic joint-affections with endocarditis.

Facial paralysis may occur as the result of prolonged otitis.

Psychoses, such as melancholia and mania, have been noted, similar to those following typhoid fever or pneumonia.

Otitis may remain with a permanent discharge and consequent deafness or mutism.

The **diagnosis** of scarlet fever in most cases presents few difficulties; but, on the other hand, there is no disease in which the symptoms are more indefinite at times. This is particularly the case with those patients who present an evanescent or partial exanthema and only slight febrile disturbance. In some cases the diagnosis must always remain in doubt. Under these conditions it is better to err on the safe side, and to take all precautions of isolation. The exanthema if partial or not very well marked is likely to be overlooked. The angina, which is the most constant symptom, may be mild. The temperature presents nothing typical as in typhoid fever.

It is good practice in the presence of a localized exanthema of a punctate character on the thighs or lower abdomen or the upper part of the chest, with angina and a slight febrile movement, to consider the case as one of scarlet fever. In all cases of sore throat it is wise not to omit an inspection of the general surface. Although some authors have described the angina of scarlet fever as typical in color, the author has never found this sign of value. In some cases of scarlatinal angina the throat is intensely red; in other cases it is of a pale-pink hue; in still others the throat is only slightly inflamed.

The **enanthema** is not of any service in making a diagnosis. The eruption on the soft and on the hard palate is not characteristic.

Albumin in the urine is thought by some to be diagnostic of scarlet fever. There may be marked and unmistakable symptoms of scarlet fever without albuminuria. A simple lacunar amygdalitis may cause it.

We must differentiate the eruption of scarlet fever from that of measles and rötheln, from drug eruptions, and those due to irritants.

Measles.—In some forms of scarlet fever the eruption on the forearms has a blotchy appearance. Near the wrist-joint the author has seen it closely resemble the eruption of measles. In these cases the punctate character of the eruption elsewhere on the surface, and the presence of angina, will assist us, in the absence of any enanthema on the buccal mucous membrane, in coming to a conclusion. In measles the diffuse localization of the exanthema on the face, the conjunctivitis and bronchitis, will aid us. In scarlet fever parts of the face, such as the alæ nasi and the region of the mouth, are free from eruption, while in measles these localities are affected by the exanthema.

Rötheln.—Scarlet fever is most frequently mistaken for rötheln, and *vice versa*.

In *rötheln*, when the eruption is punctate, it is invariably discrete. There is never the severe dermatitis with swelling of the skin found in scarlet fever. In rötheln the lymph-nodes are more constantly and generally swollen behind the sterno-mastoid, in the axillæ and groin. The throat is but slightly reddened. Rötheln presents a normal temperature or at the most a temperature at the outset of the eruption of 101°–102° F. (38.3°–38.8° C.) or even 103° F. (39.4° C.), which rapidly subsides to the normal, although the exanthema may be spreading.

Drug Eruptions.—Following the administration of quinine some children, like some adults, develop an eruption which closely resembles that of scarlet fever. In the presence of an angina and fever it may be difficult to exclude scarlet fever. Antitoxin of diphtheria, antipyrin, and belladonna also cause a rash closely resembling that of scarlet fever. It is well in such cases to discontinue. the drug, and after a few days, the eruption having disappeared, to administer it again. If the patient be susceptible, there will be a repetition of skin symptoms. Kerosene rubbed on the surface will cause a punctate eruption the exact counterpart of a scarlet fever eruption. Among the poorer classes, with whom petroleum is popular as a general remedy, we should think of the possibility of its having been used. If that has been the case, the skin will have a distinct odor of kerosene.

Prognosis.—The prognosis in scarlet fever depends largely on the character of the epidemic and the prevalent type of the disease. In some epidemics in New York City the mortality has been exceedingly low—2 to 4 per cent. (J. L. Smith), while in others it has been notably high. In England the mortality varies from 13 to 40 per cent.

Personal idiosyncrasy will affect the prognosis. Some children develop malignant septic types of the disease although the prevailing epidemic is mild.

Cases complicated with severe angina of a septic character do badly from the outset.

Nephritis is a complication greatly to be feared. It may result in uræmia and death, or the acute may be followed by a chronic nephritis which will ultimately prove fatal.

Otitis may cause serious and even fatal complications, such as brain abscess or sinus thrombosis.

Affections of the endocardium or pleura may prove fatal.

The prognosis of the so-called scarlatinal rheumatism is good. The joints, even if synovitis develops, retrograde as a rule to the normal in from two to three weeks. This may result even if high fever persists for some time during the joint-affection. In the presence of joint-complications it is necessary to be on the lookout for endocarditis or pericarditis. The occurrence of the latter takes place, as a rule, in cases in which there are other signs of septic infection, such as pleuritis and even peritonitis. All these are cases of mixed infection. If synovitis is complicated with such a serious inflammation as pericarditis, the latter is very likely to be purulent, and in that case the prognosis is grave.

We should never pronounce the patient out of danger until the fourth week of the disease has passed without serious complications. A very high temperature at the outset is an element of danger, although not necessarily so. Septic cases with high temperature and pulse above 150 in the first week of the disease are always to be regarded with apprehension.

Lotz shows that the mortality is greatest under the age of one year and between the first and second year. The lowest mortality according to statistics occurs between the tenth and the fifteenth year.

Morbid Anatomy.—**Skin.**—The investigations of Preobrachensky show that during the interval from the third day to the fourth week certain changes occur in the skin. These consist chiefly in an erythematous inflammation of the papillary layer, with hyperæmia, hemorrhages, and a diapedesis of erythrocytes and leucocytes. There is an œdematous infiltration of the connective tissue of the skin. The cells of the rete Malpighii show vacuolization. There is also an infiltration of the sudoriparous and sebaceous glands with small round cells. The epithelium of these glands desquamates and necroses. At the time of the eruption streptococci are found in the skin, especially in the vesicles of the sudamina.

The changes in the kidneys will be considered in the chapter on Diseases of the Kidney.

Bacteriology.—The parasitic nature of scarlet fever is still a matter for study. Streptococci play a leading rôle in the disease. Micro-organisms have been described in the blood (Hallier, Klebs, Tschamer). Others have seen plasmodium-like protozoa in the blood (Pfeiffer, Doehle).

Streptococci have been found in the throat membranes (Löffler), in the joints (Litten, Heubner, Koplik, Van Arsdale), and in various viscera (Fränkel, Freudenberg). Streptococci have also been found in purulent foci of the joints and pleura (Raskin), and in the kidneys, in cases which have succumbed to fatal nephritis (Babes). In septic forms of scarlet fever these streptococci exist in the circulating blood (Babes, Lenhartz, Feer). Streptococci have also been found in the cerebrospinal fluid and bone-marrow (Baginsky). Bacteriologists, however, are not willing to assign to these streptococci anything but a secondary rôle, because they present no features which distinguish them from ordinary Streptococcus pyogenes. Kurth found that some of the streptococci, the so-called conglomerate-forming streptococci, were of a virulent type. Bretonneau, Henoch, and Heubner have always distinguished the diphtheria of scarlet fever from true diphtheria. Sörensen describes the membranous formations of scarlet fever as milky, yellow, smeary deposits which cannot be peeled from the parts. The membrane seems to penetrate into the mucous surfaces. Ulcers form, and the tonsils, soft palate, uvula, and nasopharynx become a necrotic, sloughing mass. Scarlatinal diphtheria is pre-eminently an inflammatory process with high fever, swelling of lymph-nodes, and suppurations in different parts of the body. If the larynx and trachea are affected, the bronchi rarely become involved. The contrary is true of Löffler diphtheria. In the latter the membrane can be peeled from the surface of the mucous membrane. The membrane is rich in fibrin, and spreads more on the surface and not in the depths. True diphtheria is followed by paralyses. A peculiarity of scarlet fever is that it may occur sporadically for years and yet not become epidemic. This is in contrast to what occurs in measles. In the latter disease the affection may disappear almost completely and suddenly reappear in epidemic form (Henoch, Johanessen, Feer). Epidemics of scarlet fever are less common than those of measles.

Treatment.—**Prophylaxis.**—The diagnosis of scarlet fever once made, the patient should be isolated from the rest of the family. If several children are affected in the same family, these children should be separated and not placed in one room. Otherwise reinfection will occur. The clothes worn just prior to the illness should be sterilized in steam and then aired in the sun. Sufferers with angina who have been about the patient should not be allowed to come into contact with the healthy. All the children of the family should be kept from school. During the illness the bedclothes and linen of the patient should be put into a 1 : 5000 solution of mercuric chloride, prior to being boiled and dried and aired in the sun. The sick-room must be kept well ventilated. There is no advantage in keeping the temperature of the sick-chamber too low. The author has found a temperature of 68° F. (20° C.) comfortable for the patient and those about him. Sunshine and fresh air are of more value than a room uncomfortably cool. If possible, it is well to spray with some simple cleansing solution morning and evening the throats of any children of the family who are not affected.

The physician should take off his coat and vest and put on a linen robe of some kind before entering the sick-room. On his departure he should leave this robe outside the sick-room, or, better still, outside the window of an adjacent room. If the physician wears a beard, he should wash his face in a 1 : 2000 solution of mercuric chloride after leaving the patient. The hands should also be scrupulously disinfected. When he returns home he should make a complete change of clothing before visiting other patients. Carpets and superfluous furniture should be removed from the sick-room. The hanging of sheets wet with disinfectants in the door of the sick-room is not essential.

Those about the sick should have no intercourse with the healthy, nor should they go through the house. Meals should be carried by others to some neutral spot.

After convalescence the question of the disinfection of the sick-room and its occupation by others arises. It must be confessed that at present we are in possession of no absolutely sure method of disinfecting a room after its occupancy by a scarlet fever patient. We may adopt one of two methods. The cracks and spaces in the windows and doors are closed with strips of paper glued over them. The disinfectants, preferably a large quantity of binoxide of manganese, table salt, and sulphur, are placed in the centre of the room. The sulphur is then ignited and the doors sealed. Formalin is also effective. After twenty-four hours the room is opened and aired, and the floors and walls are scrubbed with 1 : 2000 corrosive sublimate. In hospitals the scrubbing is sufficient. The floor and walls about the bed occupied by the patient are scrubbed, and also the bed. The mattresses are steamed in a sterilizer constructed for the purpose. In families it is best to destroy or burn all bedding of hair. Rugs may be aired and disinfected by steam at the establishments equipped for the purpose.

How soon may a scarlet fever patient have intercourse with the healthy? We have no exact data on this important point. Some authors advise that after the termination of desquamation the patient be given a bath of 1 : 10,000 corrosive sublimate, and then allowed to mingle with the healthy. Others (Baginsky) advise prolonged isolation. It is not always practicable, nor indeed desirable, to isolate a patient for too long a period. Family considerations demand a return to the family circle as soon as possible. In these cases the course first mentioned is the most practicable. In cases which have exhibited a malignant septic form of the disease the author would advise prolonged isolation after convalescence, for the safety of the other children. The urine of a scarlatinal case if there are even mild signs of nephritis, such as albumin and casts, is believed to be infectious. A recent otitic discharge is thought to be capable of conveying the scarlatinal poison.

The treatment of scarlet fever is largely symptomatic. In an ordinary mild case there is little to do but to regulate the diet, and keep the nose and throat freed from excess of secretion. The skin needs little care. During desquamation it is anointed once a day with a 1 per cent. salicylic acid or boric acid ointment. The urine should be examined daily, for even in the mildest cases severe nephritis is apt to intervene. Vigilance should not be relaxed until after the fourth week. The fever in simple cases needs only the mildest measures. We should remember that the tendency of the fever is to mount until the eruption is fully developed. It then naturally remits. Thus a temperature of 105° F. (40.5° C.) in an ordinary uncomplicated case may not last more than a few hours. In ordinary cases sponging with lukewarm water is efficacious. The aim is not so much to reduce the temperature as to support the nervous system and the heart. In private practice it is well not to resort at once to full baths simply because the temperature is above 104° F. (40° C.). The reverse is true with temperatures which are persistently high for days. In such cases the author resorts to full baths. The patient is placed in a bath at 100° F. (37.7° C.), and the water cooled to 85° F. (29.4° C.). With children it is well not to resort to lower temperatures. This is especially true in the asthenic forms of sepsis. The patients fail to react after the bath, and seem weakened by the excessive cold. The patients remain in the bath about five minutes, and are then taken out. In cases in which the temperature mounts above 105° F. (40.5° C.) we may employ the pack at a temperature of 70° F. (21.1° C.), with much benefit if the reaction is good. The trunk pack may be repeated every one or two hours. The baths above described may be given every four hours. While the patients are in the bath reaction may be promoted by mild friction. Patients with scarlet fever, especially young children, do not bear baths below 75° F. (23.8° C.) well. The old theory that kidney complications are caused by cold baths is not proved. On the contrary, in uræmia Kussmaul lays much weight on the beneficial effects of cold packs where hot baths produce untoward symptoms (Baruch).

Antipyretics are of little value in scarlet fever, and should not be used unless there is some special contraindication against hydrotherapy. Antipyretics of the coal-tar series especially, weaken the heart in the toxæmia which accompanies scarlet fever.

Heart.—The heart is supported in septic cases with high temperature, in the same manner as in other diseases of a toxic nature. Alcohol (whiskey) is not given in mild cases. In considering its administration the kidneys should be taken into account. We wait until the temperature remains persistently high. At the third or fourth day a constant temperature of 105° F. (40.5° C.) which refuses to abate with treatment calls for the employment of alcohol with other remedies. For a child of from two to five years half a drachm to a drachm of alcohol every three hours is a sufficient dose. Alcohol and digitalis are probably our best cardiac remedies. Caffeine and camphor may also be employed. Strychnine does not seem to do so well in cases in which there is an active myocarditis.

Throat and Nose.—In inflammations of these passages we simply keep the parts sprayed with an alkaline solution in order to remove excessive secretion. In this way the patient is made comfortable and the inflammation of the fauces kept within bounds. It is not always possible to spray the throats of the little ones. If there is nasal involvement, the passages may be kept clear by syringing with salt solution. Strong antiseptic solutions or solutions of sublimate or peroxide of hydrogen are of little use if not harmful. Antitoxin of diphtheria is employed if true Löffler diphtheria coexists. In the streptococcic or most common form of pseudomembranous inflammation we have no remedy which acts directly on the inflammation. Antistreptococcic serum has not given encouraging results.

In those cases of scarlet fever in which there is great obstruction of the nasal passages and enlargement of the tonsils, with spreading of diphtheritic membrane from the tonsil to the nasal pharynx and posterior nares, there is great difficulty in breathing. It is almost impossible in some cases to cleanse the nares on account of the accumulation of secretion and pseudomembrane. The patient lies in a semi-soporose state. The lymph-nodes at the angle of the jaw are greatly enlarged This condition of affairs may set in from the very onset of the disease. In these cases the problem arises of relieving the difficulty of breathing. Any interference in a surgical way with the tonsil would be dangerous to the patient at this time. Two courses are open to us: We may intube the nostrils with soft-rubber catheter tubing, each nostril being intubed with a piece of soft-rubber catheter, extending backward toward the posterior wall of the nasal pharynx. Nos. 10 to 12 are the most available calibres of tubing. The pieces of rubber tubing are secured externally with safety-pins, being cut close to the external nares. Through these tubes the posterior nasal space can be cleansed by cautiously allowing some salt solution to run through the rubber tubing. The relief in some cases is instantaneous; in others the amount of secretion is so great as to block up the rubber tubing. There is then no other resource but to remove the tubing and to instil in each nostril 3 to 5 drops of a 1 : 4000 solution of adrenalin chloride three or four times daily. The relief from this remedy is very great in some cases. I have seen the breathing relieved at once. At the same time, owing to the fact that adrenalin is a cardiac stimulant, the patient is rather supported as well as relieved by this remedy. Its effect should, however, be closely watched. We should be very cautious in these cases not to irrigate the nostrils either too often or too forcibly, on account of the danger of ear complications, but should try every measure before resorting to irrigation. Nasal irrigation is carried out in a manner similar to that pursued in attacks of true diphtheria in the same situation.

Lymph-nodes.—The lymph-nodes, especially in the region of the angle of the jaw, are, if swollen, treated with local cold applications. This frequently affords much relief. Unless distinct fluctuation exists, we should avoid incision of the lymph-nodes of the neck. The author has seen these nodes incised at the beginning of the second week in septic cases, with very unsatisfactory results. Pus is not found in such cases, but only foci of necrosis, which are best left to nature until the patient regains strength. Later in the disease such nodes may suppurate and need incision.

Nephritis.—The treatment of nephritis is elsewhere described in detail. The lines of procedure are indicated here. Headache, vomiting, and convulsions are treated with hot baths, and by the continuous irrigation of hot saline solution (Kemp) per rectum. The kidneys are apt to be affected from the outset in malignant cases. In these cases the Kemp treatment with saline enemata is most suitable. With young or intractable children the continuous irrigation of Kemp cannot be carried out. In these cases a high rectal enema of normal saline solution (Cantani) is given twice daily or more often if necessary. If general anasarca is present, the patient is given two warm baths daily; or by wrapping him in a blanket which has been moistened with hot water and then wrung dry we may facilitate diaphoresis with hot air. Digitalis in the form of infusion is the most efficient remedy, combined with moderate doses of potassium acetate, tartrate, or citrate. Milk is the exclusive diet. Complete suppression of urine, with blood and all the anatomical elements of severe inflammation of the kidney, will sometimes be followed by an increased amount of urine. In such cases the treatment just indicated will not be efficacious. The heart must be supported, and watch kept for uræmic symptoms. Opium should be employed with extreme caution—best not at all in convulsions; chloroform inhalations with chloral per rectum are preferable. Saline enemata at 108° F. (42.2° C.), diuretin, and nitroglycerin are applicable in those cases in which there is suppression of urine.

Otitis is sometimes first indicated by spontaneous perforation and purulent discharge. In other cases pain with a sharp rise of temperature will indicate inflammation of one or both ears. Paracentesis is best performed early, even if only slight redness of the drum is present. Later in the disease (fifth or sixth week) both ears may continue to discharge profusely, with an evening rise of temperature. In some cases the author has noted slight frontal headache and drowsiness toward evening. There may be only a slight redness over the mastoid of one or both ears. It is best not to temporize in such cases, but to advise opening the mastoid process to insure drainage and avoid sinus thrombosis or cerebral abscess.

Complications in the **lung**, such as bronchopneumonia, are treated on general lines. We should in all cases be on the lookout for pleuritic effusion. Extensive effusions must be aspirated. In all forms of pleurisy, even if the amount of fluid is not large, but persists, with a rise and fall of temperature, we should introduce a needle to determine the nature of the fluid. Pus should be evacuated from the pleura in the manner directed in the chapter on Empyema.

Joints.—Joint-affections are best treated by immobilizing the affected articulations. The patient should be kept quiet, and sodium salicylate in liberal doses administered. If this is ineffectual after a few days, the joints should be wrapped in cotton moistened with oil of wintergreen, and sodium bicarbonate given in very liberal doses (grain x (0.7) for a child of three or four years, four times daily). If synovitis occurs and the fever continues high, the joint should be aspirated under antiseptic precautions, in order to ascertain if pus is present. If this is the case, an incision with drainage is the proper remedy.

II. RÖTHELN.

(*German Measles; Rubella; Trousseau's Roseola.*)

Epidemics of this disease have been described by Forney, 1784; Heim, 1812; Hildebrand, 1832; and in recent times by Thomas and Crozer Griffith. It is an acute infectious disease, contagious from person to person through the atmosphere, though not as highly so as measles. It may occur in the same person a number of times, and may attack those who have had measles. All children exposed do not develop the disease.

Age.—The youngest patient in the author's experience was seven weeks old. The affection may occur at any age. The author has met it in adults. It occurs with the same frequency in both sexes.

Prodromal Period.—There is a prodromal period, during which there may be a slight suffusion of the eyes, with swelling of the conjunctival fold at the inner canthus of the eye. In two cases observed by the author the lymph-nodes behind the border of the sternomastoid muscle were observed to be enlarged six days before the appearance of the exanthema. There is no fever or constitutional disturbance. The period of incubation is placed by Thomas and Emminghaus at from fifteen to twenty days. Just prior to the eruption there are headache, nausea, and bronchial irritation (Forcheimer, Emminghaus).

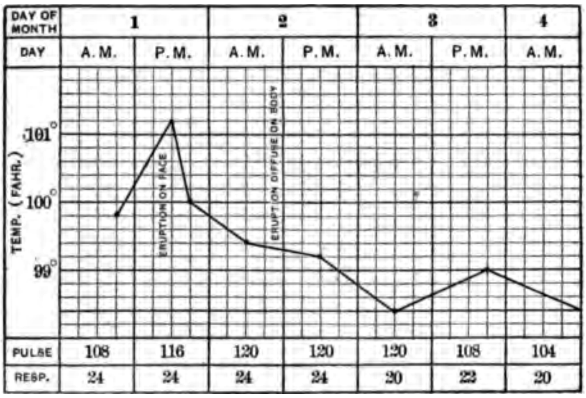
Exanthema.—The exanthema resembles that of measles so closely that at the outset it is common for physicians to mistake one for the other. It is also similar in that it is first noticed to appear faintly around the alæ nasi and on the upper lips. The exanthema appears first on the face, at the temporal regions, and on the cheeks. It is in some cases preceded by an erythematous blush diffused over the whole face (Emminghaus), which disappears in a few hours, leaving the true exanthema (pre-exanthematic erythema). The exanthema is papular, of a deep rose-red color, and distinctly arranged in crescentic outlines. This arrangement of the papules in circles and half circles can be made out where the eruption is spreading. On the face and neck it gives place to the blotchy appearance characteristic of measles. As a rule, the eruption remains discrete. Œdema is rarely present. The papules have been described as of two varieties—one the size of those in measles, and the other punctate (Thomas). The punctate papules have been seen by the aüthor on the upper part of the chest, where the eruption is confluent. They are likely to be mistaken in these cases for the exanthema of scarlet fever. In some cases of Thomas and of the author the punctate papules only were present over the whole trunk. There is an absence of the intense dermatitis seen in scarlet fever, and the individual roseolar spots have the outline above referred to. The exanthema, while fading on the face and chest, spreads slowly on the extremities. The exanthema remains discrete where it is spreading. It remains at its efflorescence on the face and trunk from a few hours to a day, when it begins to fade first from the face, and then from the trunk. A patient may present a perfectly normal skin twenty-four hours after the appearance of the eruption. Evidences of the eruption may remain on the trunk and skin for two or three days. The skin then may present bluish or brownish crescentic spots in place of the original exanthema, similar to what is seen in simple erythema. Four days after the eruption has appeared the skin in most cases will have a normal hue. There is no pigmentation or discoloration as in measles.

Desquamation.—Desquamation is not always apparent. It is possible in exceptional cases to see a very slight desquamation only at the upper part of the thorax or inner portion of the thighs.

The Eruption on the Mucous Membranes.—In rötheln the eruption on the mucous membranes does not resemble the exanthema of the skin. There is an eruption in the mouth, but it is not characteristic. There is a mild injection of the conjunctiva, a redness of the fauces, and perhaps a slight cough. Coryza, photophobia, and bronchitis are absent. The mild angina and the injection of the conjunctiva resemble what is seen in la grippe. Thomas and Emminghaus have described an irregular, spotted, streaked appearance, with small grayish miliary vesicles, on the soft and the hard palate. Gerhardt has described a spotted hemorrhagic eruption on the palate, and Forcheimer an irregular macular rose-red eruption on the soft palate. None of these is constant or characteristic of rötheln, but all are found in other affections. The *buccal mucous membrane*, however, is absolutely free from eruption of any kind, and in this fact we have a valuable diagnostic distinction between this disease and measles. In a small percentage of cases a few red stellate spots on the buccal mucous membrane have been seen by the author. In no case, however, was the measles spot with its bluish-white central speck present.

The **temperature** may at the outset be 99.8° F. (37.5° C.) in the rectum, and continue at this point throughout the disease. It may be 102° F. (38.8° C.), rarely higher. The temperature is highest at the outset when the exanthema appears on the face (Fig. 34). It falls rapidly within a few hours by a sort of crisis. Meanwhile the eruption may spread to the lower extremities.

FIG. 34.



Temperature-curve of a case of rötheln in a boy six years of age. Observed from the outset.

Lymph-nodes.—The author has observed a number of cases with especial reference to the lymph-nodes. Before the appearance of the eruption the nodes behind the sternomastoid and angles of the jaw may be enlarged. At the time of appearance of the exanthema the nodes of the axilla, bicipital groove, and groin become enlarged to the size of a bean or larger. The nodes may remain enlarged for weeks after the eruption has disappeared.

The **spleen** is not enlarged.

The Genitals.—In one case the injection of the vulvar mucous membrane caused temporary dysuria.

Complications.—Rötheln is such a mild disease that complications are rare.

Prognosis.—The patients recover rapidly.

Diagnosis.—The diagnosis of rötheln should not present any difficulties. It is most likely to be confounded with measles, scarlet fever, and erythematous eruptions.

The symptoms are much milder, and there is an absence of the specific buccal enanthema of measles. Measles does not, as a rule, present simultaneous lymph-node enlargements all over the body, such as are seen in rötheln.

Scarlet fever presents a severe dermatitis, which is absent in rötheln. There is a marked angina of a progressive type, with high temperature. The general enlargement of lymph-nodes is not so useful a sign, since in scarlet fever the lymph-nodes of the neck may be enlarged at the angle of the jaw, or those in the axillæ and in the groin may enlarge as the eruption develops. In scarlet fever there is a characteristic desquamation.

Erythematous eruptions of the small papular type may resemble rötheln, but the characteristic crescentic outline of the rötheln roseola is absent.

Treatment.—Isolation need not be rigid. Children are kept indoors in summer until the eruption has disappeared and the temperature is normal. In the winter months the patients are kept indoors one week from the onset of the disease. The angina rarely requires treatment.

III. MEASLES.

(*Rubeola; Morbilli.*)

Measles is an acute infectious disease distinguished by a characteristic eruption on the mucous membranes and skin. It is highly contagious, and is propagated through the atmosphere. The specific agent has not been isolated. Most people are susceptible to measles, and suffer from at least one attack. Infants up to the age of five months are not as susceptible as at a later period. Newborn infants have been infected by the mother, and the fœtus has been infected in utero. The fœtus in such cases may be expelled prematurely, and at birth is found covered with the exanthema; or, if the infection occurs at full term, the fœtus may be expelled alive covered with the exanthema (Squire). The firstborn only is believed by Thomas to be immune for the period mentioned. The disease is very infrequent during the first year of life. Bartels calculates the occurrence at this time at 5 per cent. of the total number of cases. The author has seen measles in infants under five months of age.

Measles is most frequent between the age of one and five years (Bartels, Henoch). It is prevalent in all countries of the globe; climate or meteorological conditions seem to have no influence upon its prevalence either endemically or epidemically.

Measles has a well-defined period of incubation, varying from thirteen to fifteen days (Van Panum). In calculating this period we include the time which elapses from exposure to the appearance of the eruption on the body. It will be seen later that this period includes the period of incubation proper, in which absolutely no symptoms, not even fever or malaise, are apparent, and the period of the enanthema on the mucous membrane. The enanthema, which may be accompanied by coryza of mild or severe type, may appear from the ninth to the tenth day after exposure, and lasts from three to five days. Thus while the coryza may be postponed several days or the enanthema may be present for a variable period, the two periods together have a duration of from thirteen to fifteen days. I have seen the enanthema fully five days before the exanthema, and have seen cases of this kind without any manifestations of coryza to signalize the onset of the disease. It is erroneous, therefore, to calculate the period of incubation from the exposure to the onset of coryza, as the latter is variable as to the time of its appearance.

One attack protects the individual from subsequent attacks. Authentic cases of two attacks in the same individual have recently been recorded. By this is not meant a recrudescence of the exanthema after it has once faded. This is also known to occur (Jürgensen). Experiments have proved that measles is highly contagious in the catarrhal stage. Inoculations with the blood (Home) and nasal secretions (Mayr) have given positive results. The period of greatest contagion extends through the period of the exanthema. It diminishes as the exanthema fades, and is thought to disappear gradually during the period of desquamation. Thus though more general in its power to infect, the poison of measles has a shorter period of life than that of scarlet fever. The poison of the latter disease may retain its power of infection months after the disease has run its course. From what has been said, it will be understood that the infection in measles takes place in the vast majority of cases in the stage of the enanthema (incubation). At this time there may be no coryza.

Infection occurs during the stage of desquamation (Baginsky). If ordinary caution is exercised, it is doubtful whether measles is ever carried by a healthy individual to a third person as scarlet fever is. Baginsky records an epidemic caused in this manner. The poison does not adhere to articles of furniture and wearing apparel with the same tenacity as in scarlet fever.

The Ordinary Type of the Disease.

The ordinary simple type of measles is that which runs its course without any complications or sequelæ. There is a prodromal period, which includes the period of incubation before the appearance of the enanthema on the mucous membrane of the mouth. During this period it is well established that there are no clinical symptoms whatever—neither fever nor malaise. At the time of the appearance of the enanthema on the mucous membrane the patient begins to feel slightly ill. The symptoms may be only a headache or a slight disturbance of the stomach. The author has noted in some cases a rise of a degree or more in temperature toward evening. There are at this time slight injection of the eyes and general lassitude. Coryza is not pronounced. The patient during the first days of the enanthema, and by this is meant forty-eight to seventy-two hours before the appearance of the exanthema on the skin, presents few signs of illness. If, guided by the very faint redness at the inner canthus of the eyes, we look into the mouth, a few spots of a very characteristic eruption are seen on the buccal mucous membrane. This eruption is pathognomonic of the invasion of measles, and will be later described as the enanthema. After forty-eight to seventy-two hours, and in some cases a longer period, there are coryza, cough, and conjunctivitis. There is a slight febrile movement, varying in intensity in different cases. The exanthema now appears, and is first noticed at the temporal region of the face and the alæ nasi as a macular rose-red spotted eruption, which becomes papular later in the course of the disease. The face and scalp are now fully covered by the rose-red irregularly shaped papules, which next appear in rapid succession on the back of the hands, forearms, anterior part of the trunk, back, and lower extremities. This order of the appearance of the exanthema is not always maintained. In some cases, as pointed out by Rehn, and verified by the author, the eruption may first appear on the back. It is, therefore, advisable to examine the patient in a nude state.

The eruptive stage of measles generally lasts three or four days, during which the patient has an exacerbation of all the symptoms of the stage of invasion. There are intense photophobia, active coryza, and a croupy cough as a result of the invasion of the laryngeal mucous membrane by the enanthema. The bronchi are also affected, and there are symptoms of acute bronchitis. Even very mild cases of measles show laryngeal and bronchial involvement. At this stage the exanthema on the skin is general and profuse, and in places confluent. The patches of healthy skin are crescentic, owing to the peculiar conformation of the papules. In some mild cases the rash may be very diffuse, but in others discrete. In the mildest forms of measles the rash closely resembles in the latter respect that seen in rötheln.

The fever reaches its height when the eruption on the skin is fully developed. If the mucous membrane is inspected at the height of the skin eruption, it will be seen that the enanthema becomes diffuse before the eruption of the skin is fully developed. The mucous membrane of the mouth is diffusely inflamed and studded with bluish-white specks which rapidly disappear or desquamate. The eruption on the skin persists for three or four days and then begins to fade. With disappearance of the eruption the general symptoms abate. The fever remits, and the temperature becomes normal by gradual morning remissions. The coryza, cough, and photophobia lessen, and the patient passes into the convalescent period. Desquamation begins when the pinkish hue of the eruption has disappeared. This stage continues until the last vestige of pigmented spots on the skin has disappeared. As a rule, it is completed two weeks after the exanthema has made its appearance. Desquamation is never absent in measles (Crozer Griffith), but it may be difficult to detect its presence. The epithelium is shed in the form of branny scales. Desquamation is best seen on the anterior part of the chest, shoulders, and inner surface of the thighs. In uncomplicated cases it is not attended by constitutional symptoms.

The Temperature.—Measles presents no characteristic fever-curve. The invasion is rarely signalized by a chill. There may be a slight sensation of chilliness. The prodromal period before the appearance of the enanthema is not marked by fever. The period of the enanthema presents a slight temperature with morning remissions to normal (Fig. 36). When the eruption appears on the skin the fever increases, and reaches its height after thirty-six hours, at the time of the full development of the eruption. The temperature continues high with morning or evening remissions for from one and a half to two and a half days, and then subsides, and disappears in from twenty-four to thirty-six hours after desquamation has set in. The temperature may reach 104°–105.8° F. (40°–41° C.) without complications. During the stage of desquamation the temperature is not elevated unless complication exists in the lung or elsewhere (Fig. 35).

I have sketched the type of disease which is not complicated by serious affection of the viscera and which has no sequelæ. On account of variations from the simple type just described, measles is one of the most dreaded diseases of infancy and childhood.

In fatal cases occurring during the first two years of life the lung is generally involved (Henoch). The appearance of the eruption is ushered in with a convulsive seizure or a chill. The pneumonia appears as the eruption reaches its height, and within two weeks either proves fatal or else leaves the patient weakened or the subject of an empyema. The infection of the kidneys may be so severe as to prove speedily fatal, or there may be severe mastoid disease. On the other hand, there are cases of measles of a type so mild as to

PLATE VII.

Fig. 1.

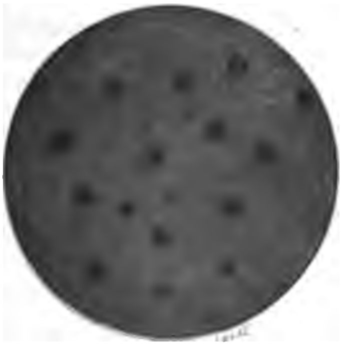


Fig. 2.

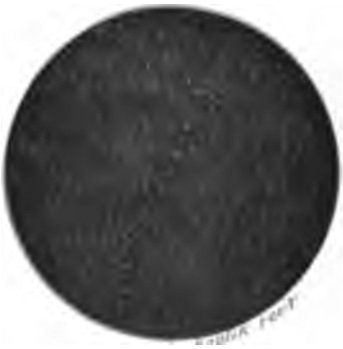


Fig. 3.

Fig. 4.

The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

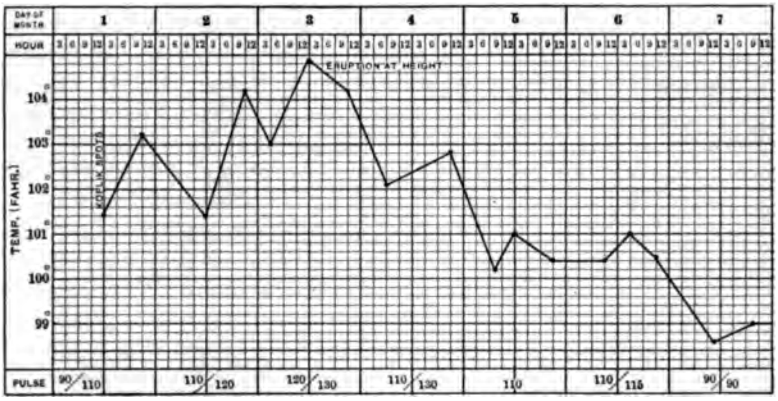
Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

cause little constitutional disturbance. The fever is very mild and evanescent, and present only at the outbreak of the eruption, and even at this stage may be so slight as to escape notice. Jürgensen records measles without fever.

FIG. 35.

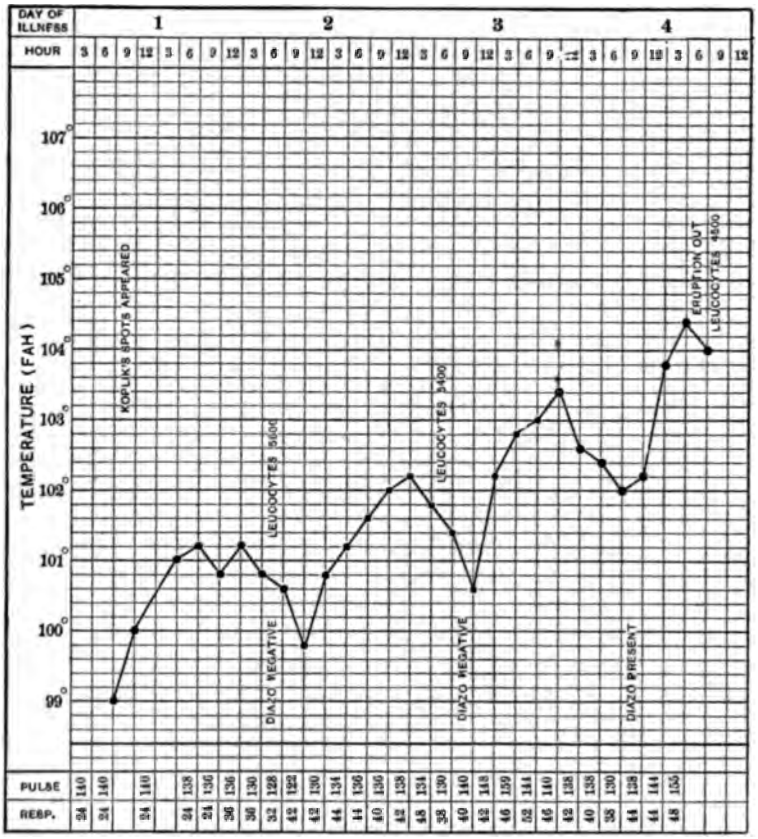


Uncomplicated measles in a boy of five years.

The Enanthema.—This is the eruption which appears on the mucous membrane of the mouth. It differs from the exanthema in respect to location. The enanthema appears in the mouth from three to five days before the appearance of the exanthema. It is accompanied by redness of the pharynx, and of the anterior and posterior pillars of the fauces. The soft palate is studded with irregularly shaped rose-colored spots or streaks. The spots on the hard palate present small whitish, punctate, miliary vesicles. These spots are also found on the otherwise normally colored mucous membrane of the cheeks and on that opposite the gums of the upper and lower molar teeth. They have been described by Flindt in these localities and on the palpebral conjunctiva. Filatow has described a desquamation of the epithelium of the mucous membrane of the lips and cheeks, in the form of minute whitish shreds (Slawyk). A complete series of studies of the enanthema of measles has been made, and there can, therefore, be no doubt of its existence. In 1896 I published a study of the enanthema on the *buccal* mucous membrane, and on the inner surface of the lips. In this study I showed that the enanthema on the hard and soft palate so frequently described since the publication of Rehn was not peculiar to measles. The spots of rose-colored papules or streaks with the superimposed miliary vesicles are found in rötheln, scarlet fever, and some cases of simple angina. The eruption on the *buccal mucous membrane* alone, however, preceding the appearance of the exanthema on the skin by a period of from three to five days, is characteristic of the invasion of measles. It is pathognomonic of the disease, and occurs in no other known conditions. It is almost invariably present, observations having shown it to be absent in only a very small percentage of cases.

On looking at the mucous membrane lining the cheeks (buccal) in strong sunlight, a very characteristic eruption of irregular stellate or round rose-colored spots is seen. In the centre of each spot there is a bluish-white speck. This appearance of a bluish-white speck on a rose-colored background is pathognomonic of the onset of measles. The speck is sometimes so minute that strong sunlight is necessary to render it visible. The number of specks

FIG. 36.



Case of measles observed from the first appearance of the "Koplik spots" to the time of the outbreak of the first signs of an exanthema, a period of fully four days. During this time it appears there was a gradually rising curve of temperature without any exanthema.

at the outset may be less than half a dozen. In a short time they become more numerous, and the rose-colored spots become confluent, so that there are diffusely red patches of buccal mucous membrane studded with bluish-white specks. The specks rarely or never become confluent; their color does not resemble that of sprue, nor are they as coarse as sprue accumulations. They are seen on the inner surface of the lips, and are sometimes well marked on the buccal mucous membrane adjacent to the gums of the upper molar teeth. If the finger is passed over the mucous membrane, they are felt to be raised and firmly adherent. They can be rubbed off by force or picked off with forceps. As the exanthema spreads, the enanthema of the buccal mucous membrane becomes diffuse. When the exanthema is at its height and during efflorescence the eruption on the mucous membrane begins to lose its characteristics. The bluish-white specks are washed away by the buccal secretions and leave the mucous membrane diffusely reddened and raw.

By referring to the temperature-curve, it will be seen that the appearance of the enanthema is accompanied before the outbreak of the skin eruption by fever of a low type (Fig. 36). There is also at this time a leucopenia; a diazo reaction appears in the urine at the time of the outbreak of the exanthema.

The **exanthema of measles** is a characteristic eruption of rose-colored or purple-colored papules, varying in diameter from 1 millimetre to 1 centimetre, the average diameter being 2 millimetres. They are irregularly circular, or longer in one diameter than another, or shaped like a half-moon. They arrange themselves crescentically. They are at first discrete, but soon become confluent, so that large areas of skin are covered. Here and there are areas of normally colored skin. The discrete papules have a distinctly crescentic arrangement. This is seen on the thorax and thighs. As a rule, the whole face is covered with the eruption, and the skin swollen. The eruption spreads from the face and head to the back of the neck, throat, upper part of the back, chest, and back of the hands and arms. The lower extremities become affected, as well as the palms of the hands and soles of the feet. As a rule, the eruption on the skin is papular; the papules may show at their summit miliary vesicles. They may become confluent and form patches. Hemorrhages may occur in and around the papules (Morbilli hæmorrhagica). In these cases petechiæ occur in the course of the exanthema, and persist into the period of desquamation. They should not be confounded with petechial eruptions or purpura, which may appear after the exanthema has run its course. The exanthema in weakly children may be limited in its distribution and not characteristic. Henoch believes that many cases in which the exanthema does not develop in sequence, take a subsequent course which may be severe. If therefore the exanthema should first appear on the back, instead of the face, and spread thence, complications may be expected. Although complications occur with eruptions which are diffuse and very general, the severity of the eruption is no index as to the severity of the disease.

When the exanthema fades, it leaves the skin studded with dirty brownish-colored spots, which have the arrangement of the original exanthema. These pigmented areas gradually fade, and when desquamation is complete they disappear.

Measles may run its course without the appearance of the exanthema on the face. It may be ill-defined and limited to certain parts of the body. It may develop in full intensity and then suddenly fade within a few hours. This occurs in cases in which severe disturbances of the circulation alter the distribution of blood in the skin. In these cases there may be a complication of the lungs or heart, but the fading of the exanthema is not, as is thought by the laity, primarily the cause of any affection of the internal organs.

The Nose, Pharynx, and Larynx.—In very young infants severe inflammation of the mucous membrane of the nose and nasopharynx may lead to difficulties not only in breathing, but also in feeding. In these cases membrane rarely develops. If it does appear, it takes the form of a pseudomembranous rhinitis, generally of a diphtheroid streptococcic nature. Its course then may be subacute. The larynx is sometimes severely affected, so that at the height of the exanthema the patient is troubled with a harassing, croupy cough. In some cases the patient becomes almost aphonic. If there is no obstruction to the breathing, this symptom, which causes great concern, disappears. The larynx may present a pseudomembranous affection of a streptococcic nature. Gerhardt has shown that ulceration of the posterior laryngeal wall may ensue from traumatism to the larynx as a result of repeated fits of coughing. If these ulcerations cause swelling of the mucous membrane, obstruction to respiration may result. The bronchitis which is always present in such cases may cause obstruction of the finer bronchi. On account of inefficient respiratory effort atelectasis and pneumonia may result, with fatal issue.

Diphtheria may complicate measles. It may precede the eruption, or may develop at any time during the attack. In all such cases the patient has been exposed to a double infection. In one case in the author's hospital service the patient had recovered from diphtheria two weeks previous to the attack of measles. Three days after the appearance of the exanthema the conjunctiva became covered with true diphtheritic membrane. The larynx then became involved, and stenosis set in within twenty-four hours after the appearance of the membrane on the conjunctiva. The exanthema in these cases is likely to fade rapidly or become hemorrhagic. Cases of diphtheria complicated with measles are rapidly fatal, since the trachea and bronchi become involved. Fatal pneumonia supervenes. On the other hand, the author has seen a croupy cough with dyspnœa, set in three weeks after convalescence from measles. Diphtheria bacilli were found in the pharynx, and yet recovery took place. In this case no pseudomembrane on the pharynx was visible. It is not always possible to decide in a given case whether there is a simple swelling of the mucous membrane of the larynx or a pseudomembranous process. In cases with severe laryngeal symptoms, if no membrane is visible, a culture of the secretions of the pharynx should be made. The temperature-curve does not aid us. Diphtheria may run its course with a low or a high temperature. The pulse is of little assistance in making a diagnosis. There is nothing in the nature of measles which predisposes toward diphtheritic infection.

During convalescence persistent hoarseness or aphonia is not infrequently seen without other disturbances. The voice gradually returns to the normal.[1]

Bronchitis; Bronchopneumonia; Atelectasis.—A very serious complication of measles is bronchitis, which may involve the capillary bronchi, causing atelectasis and bronchopneumonia. In the stage of efflorescence the bronchitis at times becomes severe. There are found on auscultation fine crepitant râles in addition to the very coarse mucous and sonorous râles. At the end of inspiration a fine crepitation is heard, similar to that present at the beginning of pneumonia. There is also subcrepitation at the close of expiration. In these cases the constitutional symptoms are severe, if large areas of lung are involved. The dyspnœa is extreme. Although cyanosis may be present, no areas of consolidation are detected on physical examination. It is reasonable to infer that in all the cases of severe inflammation of the smaller bronchi, areas of bronchopneumonia exist. Auscultation may reveal areas of lung in which the air enters imperfectly. An attack of coughing will open up the bronchi, when air again enters these areas (atelectasis). In young infants and children this form of bronchitis is a serious complication. As a rule, it leads to bronchopneumonia.

The pneumonia which complicates measles, either in the eruptive stage or in the desquamative period, is anatomically usually of the bronchopneumonic type, although the lobar form may occur. The pneumonia is caused by an invasion of the lung tissue by streptococci from the bronchi. A bronchopneumonia may at first be difficult of detection. As a rule, however, it involves a lobe of the lung in a short time. The lower portions of the lung behind are usually first involved, although the upper lobes or middle lobe may in exceptional cases be first involved. When consolidation takes place, the area of lung involved may be as extensive as in lobar pneumonia. A pneumonic process should be suspected if the temperature in the stage of desquamation does not fall to the normal. There is a distinct rise of temperature which varies in intensity, and remits in the morning to become higher in the evening. The cough becomes troublesome, and there is also dyspnœa. In such cases the temperature alone cannot be relied upon for a diagnosis. A careful physical examination will be of assistance. Under two years of age this form of bronchopneumonia is very fatal. As a rule, pneumonia complicating measles terminates, if not in immediate recovery, in a bronchopneumonia which persists for weeks. The temperature may fall almost to the normal in the morning and in the evening rise a degree or more. In addition to the bronchopneumonia there may be pleurisy, with thickening of the pleura and purulent exudate. In some cases the upper lobe of the lung shows signs of unresolved pneumonia for weeks. Emaciation is progressive. All of these cases are not necessarily tuberculous. A tuberculous process may be engrafted on a non-tuberculous bronchopneumonia at any time by infection with tubercle bacilli. In measles there seems to be a predisposition to invasion of the lung by tubercle bacilli through the catarrhal and inflamed mucous membrane of the bronchi. We can reasonably hope for recovery in many of these cases of simple chronic bronchopneumonia. If tuberculous glands, which have been dormant before the invasion of measles exist, they form focal points for the development of tuberculosis of the lungs or meninges. Such cases are fatal. Autopsy will reveal recent lesions alongside of old tuberculous foci.

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[1] Prudden and Northrup, in a paper on diphtheria with fatal pneumonia, record 3 cases of fatal diphtheria complicating measles. The diphtheria and subsequent pneumonia were of the streptococcus variety. The 3 cases formed part of a series of 17 cases of streptococcus diphtheria followed by pneumonia.

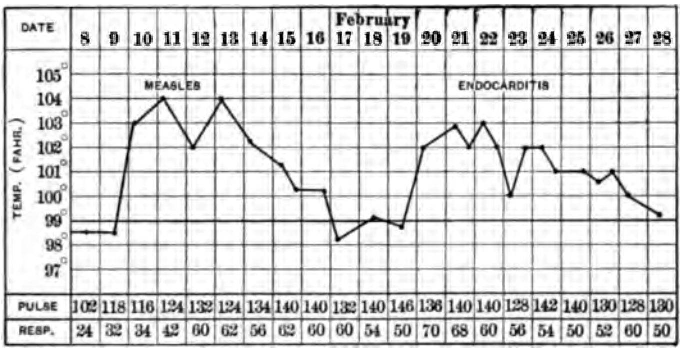
The frequency of infection with tuberculosis varies in different localities. In some epidemics it occurs in 5 per cent. of the cases; in others, 16 per cent. or more are affected (Bartels, Jürgensen).

The Heart.—The endocardium is rarely affected in measles. If endocarditis does occur, it is usually an intercurrent affection in a rheumatic subject. Fig. 37 shows a temperature-curve from a case in which rheumatism preceded an attack of measles, and which in turn was followed by endocarditis. Myocarditis may be found in fatal cases of bronchopneumonia. In bronchopneumonia complicated with pleurisy, pericarditis may also be present (Baginsky).

The Intestines.—In some epidemics diarrhœa is a frequent complication. The movements are numerous, and watery in character. When the large intestine is involved the stools contain blood and mucus, and tenesmus is present. The season of the year influences the intensity of the infection. In the warm months the diarrhœa may be of a severe type. In cases recorded by Henoch and Thomas, autopsy showed enlarged Peyer's patches and solitary follicles resembling those seen in typhoid fever. No cases of ulceration have been recorded. Jürgensen is inclined to consider the diarrhœa a result of infection of the intestinal mucous membrane. The enanthema appears in this locality early in the disease.

The Kidneys.—In many cases of measles, albumin and a few hyaline and epithelial casts are present in the urine. They are the result of a parenchymatous inflammation of the kidney, due to the poison of the disease. A true nephritis, such as is common in scarlet fever, is rarely seen. Nephritis is apt to occur in the severe cases complicated with bronchopneumonia. There may then be marked albuminuria, blood, and casts of all kinds in the urine, with suppression. On the other hand, nephritis in the stage of desquamation is rare. There is always in such cases suspicion that an infection coincident with scarlet fever may have been overlooked (Henoch). If diphtheria complicates measles, nephritis is likely to be present.

FIG. 37.



Measles complicated with endocarditis in a boy six years of age.

The Bones and Joints.—The author has seen osteomyelitis with suppuration of the joints follow measles. Streptococci were found in the pus. In one case bronchopneumonia was an earlier complication. These cases are rare.

Lymph-nodes.—If the inflammation of the throat is severe, the lymph-nodes at the angle of the jaw and underneath the body of the jaw may be enlarged. Rarely, however, is the adenitis as severe as in scarlet fever. The glands or nodes in the axillæ, bicipital groove, over the internal condyle of the elbow-joint, and in the groin may be enlarged to the same extent as in rötheln, as a result of the processes taking place in the skin. Severe infection of the gut may cause swelling of the mesenteric lymph-nodes, which, if not tuberculous, will retrograde after the disease has run its course.

The Blood.—In measles as distinguished from scarlet fever an examination of the blood shows a subnormal number of leucocytes or a leucopenia. This condition of the blood is found in the initial stage of the disease, and persists well into the period of the exanthema in uncomplicated cases, as is well shown in Fig. 36.

The Nervous System.—It is rare to see convulsions usher in an attack of measles, even of a severe type. In anomalous forms of the disease complicated with pneumonia there may be cerebral symptoms similar to those seen in the latter affection. There may in some cases be a complicating cerebrospinal meningitis with purulent exudate. If tuberculosis is present, the meninges may be attacked, as in any tuberculous infection. French writers have observed neuritis following measles.

The Eyes.—Following severe cases of measles, photophobia, spasm of the orbicularis, inflammation of the lachrymal duct, conjunctivitis, ulcerations of the cornea, and amaurosis may result. Hence, even in mild forms of the disease the eyes should be frequently inspected (Eversbusch).

The Genitals.—The author has seen dysuria in cases in which the enanthema affected the mucous membrane of the vulva in girls. Henoch records cases of gangrene (noma) of the genital organs.

The Mouth.—Inasmuch as the mucous membrane of the mouth is the seat of an active eruption, stomatitis is likely to be present, especially if through carelessness or traumatism the mucous membrane has become infected with bacteria from without. In such cases aphthæ may result. Children in unhygienic surroundings are likely to develop noma of the cheek if exposed to the infection.

Pertussis as a complication of measles is occasionally found. As in diphtheritic infection, there must have been exposure to the contagion of both pertussis and measles, since etiologically the diseases have nothing in common. The danger in the coincident occurrence of measles and pertussis is that bronchopneumonia is likely to develop, and prove a serious if not fatal complication.

The Ear.—The external structures of the ear may be affected by œdema and swelling. The external auditory canal may become the seat of painful swelling and diffuse inflammation. Gangrene of the pinna has been noted (Nottingham, Bourdillot). The most common affection of the ear is otitis media catarrhalis. Of 33 cases of severe complicated measles, Tobeitz found otitis of this variety in 16. The frequency of otitis varies with different epidemics. The otitis makes its appearance in the period between the seventh and the twenty-sixth day following the development of the exanthema. Of 22 fatal cases of measles, otitis was found in 19, only 7 of which presented symptoms during life. The great majority of cases of otitis give no pronounced symptoms and end in resolution. These mild cases are the result of the action through the blood of the measles poison on the ear structures (hæmatogenic). The severe cases follow a mixed infection through the pharynx and Eustachian tube. In the pus of acute or chronic otitis, with or without inflammation of the mastoid, the streptococcus, Staphylococcus pyogenes, and pyogenic diplococci have been found. The general course of otitis is not so severe as that of scarlet fever. In some epidemics the severe and fatal cases are more common than in others.

Sequelæ.—Any of the complications named above may pursue a chronic course. In this sense only are they sequelæ. Chronic blepharitis, blennorrhœa, keratitis, otitis, catarrhal inflammation or ulceration with stenosis of the larynx, septic retropharyngeal abscess, and chronic bronchopneumonia may persist for weeks or months.

The **prognosis** in measles varies with the virulence of the epidemic, the resistance of the individual, and the age of the patient. It is certain that the idea prevalent among the laity, that measles is a comparatively mild affection, is incorrect. In the cases treated in both dispensary and private practice, and at all periods of infancy and childhood, the mortality is 8 per cent. (Breyer). The mortality is greatest during the first year of life, and may vary in different epidemics from 10 to 40 per cent. The lowest mortality seems to be between the fifth and eighth years—6 per cent. (Baginsky). Hospital statistics are of little value to the general practitioner, as the class of cases treated in these institutions give a high mortality-rate. The mortality in hospitals may be as high as 30 to 35 per cent. (Henoch, Fürbringer).

The **diagnosis** will in most cases present few difficulties if the physician follows a fixed routine in the examination of the patient. The mode of onset, the coryza, the enanthema of the buccal mucous membrane, and the skin eruption are characteristic. If the physician will examine the inner surface of the cheeks and the buccal mucous membrane in every seemingly slight indisposition of children, he will in certain cases be able to predict an attack of measles far in advance of the appearance of the exanthema. In some cases the enanthema appears on the buccal mucous membrane before coryza is present. The inspection of the buccal mucous membrane thus becomes important as a prophylactic measure. Strong sunlight is essential for thorough inspection. Although the bluish-white spots on the rose-red background may sometimes be seen by artificial light, especially electric light, a diagnosis of measles should never be made at night. Cases of influenza closely resemble measles at the outset. These present the injected conjunctivæ, cough, and rose-colored spots on the soft and the hard palate seen in measles. In la grippe, however, the buccal mucous membrane is pale and presents absolutely no eruption. In one of the early grippe epidemics in New

York the children showed an ill-defined roseolar eruption on the surface, but the buccal eruption was never present.

Rotheln in some cases resembles mild measles so closely that the author has often questioned whether so-called cases of mild measles without rise of temperature, described by authors, were not cases of rötheln. The difficulty in differentiation is increased if measles is prevalent at the same time. The absence of the buccal eruption is a crucial test. Schmid has also laid stress on this point. In some rare cases of rötheln there may be seen an isolated rose-red spot here and there on the buccal mucous membrane, but the bluish-white speck in the centre of these spots is never seen as in measles.

Scarlet fever may at times closely resemble measles, especially in those forms in which the eruption on the face is evanescent. In scarlet fever the buccal mucous membrane has a normal hue. The author has seen scarlet fever complicated with measles. In these cases the scarlet eruption appeared first. Within two or three days there was a general recrudescence of the exanthema, with the appearance all over the body of a roseola (the scarlet rash had faded somewhat), coryza, and the buccal eruption. In other cases the scarlet fever eruption on the back of the hands and forearms assumes the blotchy, papular roseolar form of the exanthema of measles. The author has seen a case of this kind in which an expert entertained the possibility of rötheln or measles. The buccal enanthema was absent. The subsequent course of the case proved the diagnosis of scarlet fever to be correct.

The roseola of typhoid is sometimes so abundant as to mislead the physician into mistaking it for the eruption of measles. Measles complicating typhoid at the end of the second week has come under the author's notice. In this case the buccal eruption was profuse.

Antitoxin and drug eruptions may simulate a measles eruption, but the buccal mucous membrane never presents the enanthema.

The roseola of syphilis frequently resembles that of measles so closely as to cause uncertainty in the diagnosis. Here the conjunctivæ may be injected, and there may be a slight febrile disturbance (Sobel). The buccal mucous membrane is pale, and shows no eruption resembling that seen in measles.

The diagnosis of measles thus resolves itself into a recognition of the disease before and after the appearance of the skin eruption. Before the appearance of the eruption there is very little to guide us. Cough, coryza, and fever may accompany an influenza. In these cases the buccal eruption is of great diagnostic value. After the eruption appears, the question narrows itself to the differentiation of measles from rötheln or scarlet fever, and the recognition of the various forms of erythema, roseola, drug and antitoxin eruptions.

Treatment.—Prophylaxis.—As soon as the physician has made the diagnosis of measles or suspects its presence, the patient should be isolated from the other children of the family. Among the poor it is sometimes impossible to do this. The members of the family not directly concerned in the care of the patient should be denied admittance to the sick-room. It is not necessary to cover the door of the room with cloths or sheets moistened with disinfectants. The physician before entering the room should take off his coat and put on some convenient linen gown or bath-robe, so as to completely cover his person. This robe should hang outside the door of the room, so as to be easily accessible. When not in use, it should be hung in the open air. If the physician wears a beard, he should wash it after leaving the patient, for if the patient coughs in the physician's face, he is likely to carry the infection in his beard to the next child visited. Should the measles be complicated with diphtheria, extra precaution is necessary.

General Treatment.—A typical mild case of measles needs little medicinal treatment. We try to make the patient comfortable. The temperature of the room should be about 68°–70° F. (20°–21.1° C.), if possible. The ventilation should be constant and attained by means of opening doors and windows of rooms communicating with the sick-room. It is not necessary to darken the room very much; in fact, Bartels has shown that light and air are necessary to the comfort and well-being of the patient. The author has found that the ordinary yellow window-shade, if drawn over the windows, sufficiently excludes the actinic rays which are irritating to the eyes.

In a typical case of measles a temperature of 104°–104.5° F. (40° C.) may be ignored. It should be remembered that the fever continues only during the period of the eruption. With the fading of the exanthema the temperature becomes normal. It is only in cases in which there is a high temperature with delirium that medication is called for. It is not uncommon to see children covered with an eruption and with a temperature of 104° F. (40° C.) playing in bed with toys.

The cough will sometimes need treatment. In such cases I am accustomed to prescribe ♏ iv (0.25) of paregoric combined with ♏ ij (0.12) of syrup of ipecacuanha, every three hours. If the patient is kept awake by the cough, a small dose of Dover's powder (grains j or ij) (0.06 or 0.12) or codeine (grain $\frac{1}{10}$ to $\frac{1}{8}$) (0.006 to 0.008) at night will be sufficient. If the patient is very restless at night and we do not wish to give opiates, grains v (0.3) of trional will quiet a child of five years. Some young children can be put to sleep by a small dose of phenacetin (grains ij) (0.1). In a mild case, especially if there is pruritus or irritation of the skin, there is no objection to sponging the patient once a day with water at 100° F. (37.7° C.), containing some alcohol or a pinch of sodium bicarbonate.

The food should be light. Milk, broths, and, when the fever has defervesced, chicken, soft-boiled eggs, jelly, toasted bread, crackers, rusk (*Zwieback*), and cereals in attractive form, with cocoa, comprise the diet list. Orange-juice or weak lemonade may be given in moderation. Water-ices may be given, if desired.

As soon as desquamation has set in, I direct the body to be anointed every second day with an ointment of washed benzoinated lard combined with 5 per cent. of boric acid. The patient is allowed to get out of bed as soon as the temperature has fallen to normal, and is permitted to go out of doors three weeks after the outbreak of the eruption in the summer and four weeks in the winter months. Before mingling with other children, the patient should be thoroughly washed with soap. It is not necessary to put an antiseptic in the bath.

The Treatment of Complications.—BRONCHITIS; BRONCHOPNEUMONIA.—A severe inflammation of the finer bronchi is likely to cause as much fever, dyspnœa, cough, and restlessness as a primary bronchopneumonia. The temperature then rises and continues elevated—104°, even 105° F. (40°–40.5° C.)—with morning remissions. In these cases the temperature must be reduced. I never hesitate to utilize hydriatic measures. The most convenient mode of applying water is by means of compresses moistened with water at 80° F. (26.5° C.). If the patient reacts well, the compresses may be applied at 67° F. (19.4° C.); if he becomes cold and cyanosed, at 105° F. (40° C.). These warm compresses are at times very soothing, causing the patient to drop into a quiet sleep. It should be remembered that the object of applying the compresses is not always to reduce temperature rapidly, but rather to stimulate the heart and support the patient. Douching the head with ice-cold water, as recommended by some, is a very questionable practice. The use of the coal-tar antipyretics should be avoided. In lowering the temperature they act as depressants. In severe cases of bronchopneumonia aconite should not be used to lessen the rapidity of the pulse. Caffeine, camphor, strychnine, and digitalis in proper doses are more satisfactory. If a bronchopneumonia be prolonged into the convalescent stage, we should be on the alert for pleuritic effusion. This is especially likely to occur if the pneumonia lasts longer than two weeks. In these cases the symptoms present are similar to those described under Pleurisy, and the treatment is carried out on the same principles.

The LARYNGEAL SYMPTOMS become harassing when there is much swelling or slight erosions of the laryngeal mucous membrane. In such cases an improvised tent should be erected over the crib or bed and filled with steam vapor saturated with thymol or turpentine. Older children can be persuaded to breathe the vapor generated in an open kettle. If symptoms of stenosis appear, it must at once be determined by culture whether a diphtheritic process, a streptococcic pseudomembranous formation, or a stenosis due to simple catarrhal œdema of the mucous membrane is present.

DIPHTHERIA.—Antitoxin is indicated in diphtheria either of the conjunctiva, pharynx, or larynx. A large dose should be given at the outset, on account of the virulent nature of this affection as a complication of measles. We should not be too ready to intubate on the first appearance of stenotic symptoms. Many of these cases improve. The introduction of a tube into the inflamed larynx in measles is not without danger of causing ulcerations of a troublesome type after the measles has run its course. It is well to follow O'Dwyer's advice in such cases—withhold the tube as long as dangerous dyspnœa is absent. The use of apomorphine, tartar emetic, or turpeth mineral, so popular with continental physicians, to expel membrane or secretion, is of doubtful value.

THE EAR.—Otitis should be suspected if there is restlessness and an intermittent course of temperature without apparent cause. Older children may indicate the seat of pain. In some cases it may be necessary to incise the tympanic membrane. The procedure affords relief from pain, and is without ill effects. Pus or a few drops of serum only may be evacuated.

DIARRHŒA requires the same treatment as a primary enteric catarrh.

The care of the EYES, NOSE, and MOUTH should be conducted on general lines. If the secretion is excessive, the eyes may be bathed once a day with a lukewarm weak saline solution. Unless the secretions are excessive, the nostrils should not be syringed or douched. If clots of mucus or pseudomembranous shreds form in plugs, they may be dislodged once a day by a nasal washing with a suitable hand syringe. The mouth should not be washed more than once a day. This should be done both for infants who are fed artificially and for older children. On account of the great vulnerability of the mucous membrane in this disease the utmost gentleness should be exercised lest aphthous ulceration be developed.

IV. VARICELLA.

(*Chickenpox*; (Ger.) *Windpocken.*)

Varicella is an acute infectious disease with a characteristic exanthematic eruption. It is distinct from vaccinia or variola, is an affection of childhood, occurring before the tenth year, rarely later, and is transmitted through the atmosphere. It cannot always be conveyed by inoculation, as is the case with vaccinia or variola. It does not protect from vaccinia or variola. Varicella, vaccinia, and variola have been observed to attack the same patient successively at very short intervals. Few children escape after exposure, and one attack does not confer immunity. Varicella is an endemic disease, and does not occur epidemically.

Incubation.—Varicella has a period of incubation during which competent observers have noted no disturbances (Henoch); others record malaise, coryza, and sore throat. The author is inclined to regard the prodromal period as free from symptoms. The period of incubation is usually fourteen days, but it may be protracted for nineteen days.

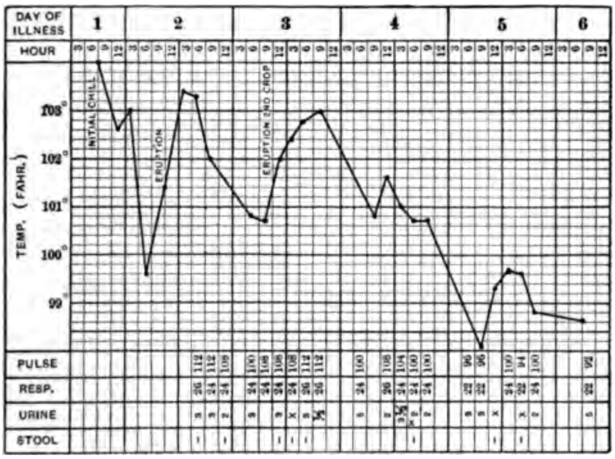
The **symptoms** consist of an exanthema, an enanthema, fever, and slight malaise. There may be complications. Previous to the appearance of the exanthema there may be a slight febrile movement and malaise, which in children may pass unnoticed. In cases pursuing a normal course, a chill with a marked rise of temperature may precede the eruption by fully twelve hours. When the eruption appears the temperature gradually falls, unless another crop of papules appears, when there is another sharp rise of temperature. Sore throat and slight malaise may herald the eruption. Previous to the appearance of the rash there may be, as in measles and in varioloid, an erythema of the surface prior to the appearance of the exanthema.

The **exanthema** consists of an eruption of roseolar papules varying in size from that of a pin's head to that of a split pea. They first appear on the forehead and face, and spread to the trunk. In some cases larger blotches appear, but these are of the nature of an erythema, which may precede the eruption of the roseola by a few hours. The roseolar papules have a characteristic violet-rose tint, are raised above the surface, and are sometimes hard to the touch. In a few hours the papule develops on its summit a vesicle, which rapidly fills with lymph. These vesicles become tense, and if the papule is irregular in shape cover the whole upper surface of the papule. In many places the vesicle at the stage of its efflorescence presents an umbilication which strongly resembles that seen in the vaccinia pock. The contents of the vesicle become cloudy and then yellow; the vesicle is surrounded by a dusky pink areola. In the course of a day or two the cycle is completed, and the vesicopustule begins to desiccate. A reddish-brown scab is developed. Many of the roseolar papules do not develop the vesicle and pustule. While one crop of papules is going through the cycle described above, others appear on various parts of the body. It is characteristic of varicella to have the surface covered with roseolar papules, papules with vesicles, and with pustules, in various stages of development. The papules vesicles, or pustules may be few or very abundant. In some cases after the scab of the vesicle has fallen off a distinct scar is left, similar to that seen in vaccination, but much smaller; it may persist for years. The skin between the papules and vesicopapules is normal in color.

The soft palate and sometimes the hard palate may show a few isolated papules, vesicles or vesicopustules similar to those seen on the cutaneous surface (enanthema). In most cases there is an angina, an injection of the conjunctivæ or even an enanthema on the ocular conjunctiva (Henoch). Thomas records varicella papules and pustules on the nasal and vulvar mucous membrane (Fig. 38).

The temperature is in many cases little raised above the normal. In others it reaches 103° F. (39.4° C.) at the outset of the affection. In rare cases 106.5° F. (41.3° C.) has been observed. As soon as the eruption is fully developed the temperature rapidly becomes normal. The duration of the fever varies from one to three days. I have seen severe cases in which the high temperature persisted fully a week. The eruption was in these cases accompanied by secondary pustulation.

Fig. 38.



Varicella temperature-curve showing successive rises due to a new eruption of papules and vesicles. Boy aged six years.

Other Symptoms.—Many infants and children show little constitutional disturbance. In other cases there is lack of appetite with excessive irritability. In others, on account of the profuse eruption in the vulva and around the nates, there is annoying vesical tenesmus and even rectal tenesmus. The latter condition I have seen in a child two and a half years of age, in whom there was a profuse eruption of vesicles in and around the introitus vaginæ, on the nymphæ, and around the anus. There is in some cases a recrudescence of the exanthema in various parts of the body, with rises of temperature.

Complications.—Gangrene of the skin with sloughing of large areas has been noted by some observers (varicella gangrenosa).

The conclusion is inevitable that in many of these cases there must have been a mixed infection.

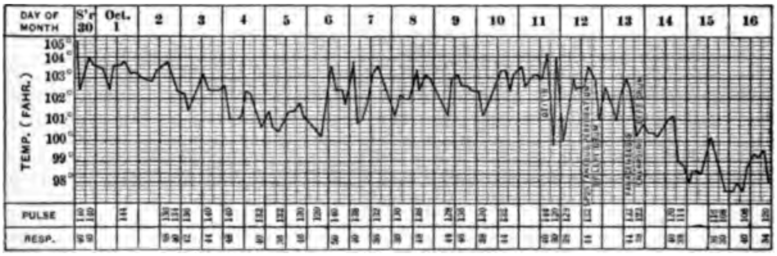
Nephritis.—In many cases there is albumin in the urine to the extent of a trace. Henoch has described 6 cases of varicella complicated with nephritis on the eighth to the fourteenth day after the appearance of the eruption. In these the eruption was profuse and accompanied by fever; there was œdema with albumin and casts in the urine. One case with fatty liver and moderate hypertrophy and dilatation of the left ventricle resulted fatally. Other authors have confirmed the observations of Henoch. I have seen slight albuminuria in some cases of varicella.

Joint-affections.—I have observed two cases of varicella with swelling, pain, and effusion in one or both knee-joints. In neither was there suppuration. Both cases retrograded, and in a few days the joints became normal. The whole picture simulated what is seen in some cases of scarlet fever. There was no endocarditis.

Otitis may occur as a complication of severe cases.

Pneumonia is an occasional complication (Fig. 39).

FIG. 39.



Varicella bullosa, pneumonia, otitis media purulenta. Female child aged six years.

The **diagnosis** of varicella should present few difficulties. I have seen a number of cases in which the eruption was not only very profuse, but the individual varicella vesicles or pustules were also very large. In these cases there may always arise the question of differentiation from the more serious affection, variola or varioloid, especially if an epidemic of smallpox is prevalent. The diagnosis may even in some rare cases remain in doubt (Jürgensen). In varicella the temperature is lower and the rise shorter in duration than in even a mild case of smallpox. In the absence of an epidemic, the mildness of constitutional symptoms, discreteness of the varicella eruption, and the absence of any œdema of the skin between the vesicles will aid us.

In some cases the eruption of roseola papules on the face and trunk has not the characteristic appearance of vesiculation or pustulation seen in varicella. It is difficult on account of the effects of the scratching of the patient to differentiate the eruption from pustules of a furuncular type. Under such conditions a close inspection of the back may result in the discovery of one or two typical varicella vesicles.

The **prognosis** is very good in varicella, except in neglected cases, in which sepsis may complicate the disease. The very rare cases of nephritis (Henoch) should be borne in mind. In private practice and in a large ambulatory clinic I have rarely seen the severer types of this disease. I agree with Fürbringer in thinking that such cases raise the question of the possibility of an extraneous infection.

Treatment.—Though the course of varicella is mild, the cases should be isolated like those of any other infectious contagious disease. We can never predict the outcome of a number of cases occurring in epidemic form, although individual cases do well. If there are itching and tension, the eruption is covered with 5 per cent. boric acid ointment applied without lint. The children are allowed out of doors as soon as the temperature has become normal, the scabs of the varicella vesicles or pustules have fallen off, and the skin has become normal.

V. VACCINATION.

Vaccination is a prophylactic measure against variola practised on the human subject. It gives a certain, though not lasting, immunity against the disease. It is accomplished by inoculating the human subject with the contents of the cowpox vesicle.

Cowpox or vaccinia (vacca, cow) is a specific exanthema which occurs on the udder of the milch cow, hence the name. Vaccinia is inoculable from animal to animal, and also on the human subject. It occurs only at the point of inoculation.

Successful vaccination gives the human subject almost certain protection for a long time against vaccinia or cowpox and variola or smallpox.

The essential cause of vaccinia in animals and the human subject has been described by Guarnieri and Kurlow as vaccine corpuscles. These are found in the vaccine vesicle and pustule. They are peculiar, finely punctate, amœbic masses of protoplasm, showing vacuoles. Loudon and Salmon, on the other hand, deny any specific properties to these corpuscles. They think they are simply degenerated leucocytes, and are seen in other simple forms of inflammation.

Edward Jenner (1749–1823) was the first to establish the doctrine of vaccination on scientific experimental data. He was the first to use humanized vaccine—that is to say, to inoculate the human subject with lymph from a cowpox vesicle, and then to utilize the lymph of the vesicle in the human subject to inoculate others. This method has been abandoned. To-day the lymph used is obtained directly from the animal. The lymph is, as a rule, inoculated from animal to animal for several generations. It is just as effective as the lymph of the first animal of the series inoculated. It is called animal lymph or vaccine. The disadvantages of using humanized vaccine are many. First, there is a natural reluctance among some people to vaccinate their children with lymph obtained from the human subject. Apart from the popular belief in the transmission of tuberculosis, scrofula, and other forms of blood disease in this way, it is not always possible to exclude an infection, such as syphilis. The animal lymph can be controlled in its manufacture and produced with all scientific precautions. Animal lymph and human lymph do not differ in the power to confer immunity against variola. The animal lymph should be obtained from the healthy animal in the vesicular stage of the eruption; this is the fourth or fifth day of cowpox. It is preserved by mixing it with three or four times its bulk of glycerin. It may be put up for use on quills or ivory slips in a dry state or in small capillary tubes in the liquid condition. The so-called vaccine pulp, made up of the contents of the vesicle and its epidermal covering, and preserved in glycerin, is not used in this country.

Age at which to Vaccinate.—Every infant and child should be vaccinated. There is no contraindication except some acute or chronic illness. Even the hemorrhagic diathesis is no contraindication. Vaccination is best done between the fourth and the sixth month, before teething has begun (Zimmerman). In an emergency, such as the presence of an epidemic of smallpox, the newly born infant may be vaccinated.

Mode of Vaccination.—Boys are vaccinated on the left arm; girls, for esthetic reasons, may be vaccinated on the thigh or calf of the leg instead. The outer surface of the arm, at about the insertion of the deltoid in the humerus, is usually selected. The skin is carefully cleansed with soap and water, washed with alcohol, and dried. With a clean sewing-needle the skin is scarified three or four times in one direction, and at right angles to the first scarifications. We should not cause bleeding, but only expose a raw surface. The scarified area should be about one-eighth of an inch square. The lymph is now rubbed on the scarified area. If quills are used, the vaccine on the quill is moistened with a drop of distilled water before inoculation. Scarifying large areas is likely to cause excessively large pustules, with subsequent severe inflammatory reaction. On the other hand, a small area of scarification may give a very large pustule. In other words, the size of the vaccine pustule does not always depend upon the size of the area of scarification. A mixed infection will give a severe reaction with a very small area of scarification.

Lymph to Use.—Either the liquid or the dry lymph may be used.

Both are reliable if recently prepared. If the lymph is not fresh, or there is carelessness in its use, the vaccination will be a failure.

Course of the Vaccination.—The great majority of vaccinations are very uniform in history. There is an incubation period, during which the wound heals. There are absolutely no symptoms. This period usually lasts three days, sometimes only two, and may be prolonged to four or six days. After this period there is the eruptive stage, ushered in by the formation of flat rose-red papules at the points of scarification. The papules are either oval or irregularly long. On the fifth day a vesicle appears in the centre of the papule and spreads to the periphery. On the sixth day the vesicle takes up the whole papule, has a pearly lustre at the surface, and presents a central umbilication (Jenner's vesicles). The seventh day is the day of efflorescence; the vesicle is filled and tense with lymph, has a rose-red areola and a hyperæmic zone outside this areola; there are itching and tension. On the eighth day the contents of the vesicle become slightly cloudy. On the ninth day the suppuration is pronounced, and on the tenth day the suppuration, swelling, and inflammatory reaction are at their height. At the end of the tenth day there is a retrogression of all the symptoms. The vaccine pustule becomes less angry looking and the inflammatory reaction subsides. A crust forms which may become dry, hard, and fall off, leaving a scar beneath. This takes, as a rule, from ten to fourteen days.

Fever in some cases begins on the fifth day after vaccination. It may be slight and reach its height between the eighth and the tenth day. There may at this time be slight digestive disturbances, such as vomiting or greenish movements.

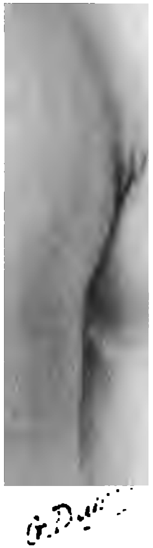
The areola around the vaccine pustule may spread so as to involve most of the upper part of the arm, or the inflammatory reaction may spread over the entire arm, and sometimes over the back. There may be enlargement of the lymph-nodes in the axillæ. These lymph-nodes may suppurate. If there has been no mixed infection, they retrograde with the pustule.

Complications.—Complications occur according to Sobel in 14 per cent. of vaccinations, and are the result of traumatism of the pustule, mixed infection (that is, the presence of impurities, such as streptococci or staphylococci in the lymph), lack of cleanliness at the time of maturation of the pustule, and retention of pus in a dressing. The most common complication is an exceedingly severe reaction, with an extensive necrosis of tissue. This may affect the fasciæ or muscular layers, causing large loss of tissue. Among the rarer complications of vaccination is a true septic infection. In these cases there is a history of mismanagement of the pustule, such as traumatism or the compression of the arm by a bandage. Infection which manifests itself in a remittent febrile curve occurs. In one case which came under my notice a few pus-corpuscles appeared in the urine, the elbow-joint and other joints became painful and swollen, and suppuration in the joints resulted. These cases are fatal. There is a true osteomyelitis of the heads of the bones, with formation of pus in the joints. In other cases the child may by scratching inoculate itself elsewhere, either on the arms or even lips and eyelids; the latter condition has come to my notice. It forms a very painful and severe complication. Erysipelas may set in early or late in the history of the vaccination. It may spread down the arm and forearm on the trunk and may endanger the life of the patient. In other cases there may be suppuration of lymph-nodes. In susceptible subjects a rebellious eczema may appear as a direct sequence of the vaccination. Among the complications may be mentioned axillary adenitis, hemorrhage into the pock (trauma), exuberant granulations, and keloid of the scar. Roseman found that the dry points contain more bacteria than glycerinized vaccine. All vaccine contains pus-organisms. He thinks that properly prepared glycerin lymph is to be preferred to dry points. The same investigator examined a large number of samples of commercial vaccine and failed to find tetanus germs in them. It seems more likely that carelessness in dressing or handling, the pernicious use of shields, or faulty technique in performing the operation has been the means of introducing tetanus-spores, rather than that these should be present in the vaccine virus.

Generalized Vaccinia.—This is a general eruption of vaccine pustules, which in rare cases appears from the third to the seventh day over the whole trunk and extremities. It is really a generalized cowpox, similar to the generalized eruption in the exanthemata. D'Espine and Jeandin describe cases in which there can be no doubt of the absence of infection of the surface by the nails or otherwise. The prognosis in these cases is good; there are no severe symptoms, and the fever is slight.

The **management** of a normal case of vaccination is important. We should protect the vesicle from traumatism by means of some simple contrivance, such as a shield. If the areola is angry looking and the redness and swelling severe, we may paint it once a day with compound tincture of benzoin. This is very soothing and protects the surface from friction. If complications occur, they should be treated on surgical principles. Above all, there should be no retention of pus by any form of dressing. Dressings which seal the vaccine pustule hermetically from the air cause retention, and are therefore dangerous. Sepsis as described above is not the result of vaccination, but of subsequent mismanagement.

Vaccination Eruptions.—The eruptions which follow vaccination or occur while the pustule is still in course of development are of interest. Sobel has made an exhaustive study of these eruptions. 2 per cent. of the vaccinations are followed by more or less gen-



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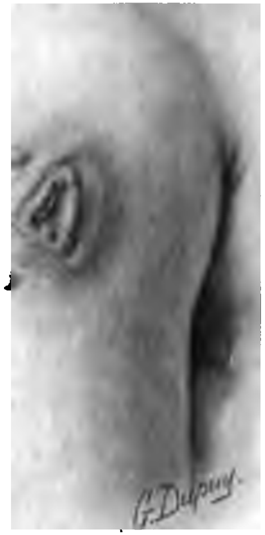


FIG 3

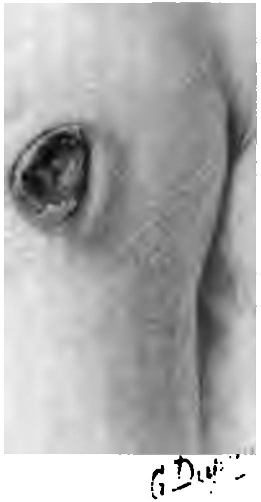


Fig. 1. Vaccine Vesicle on the Sixth Day, after Inoculation. Contents of the vesicle serous.

Fig. 2. Vaccine Vesicle on the Ninth Day, fully formed and beginning to suppurate. Secondary vesicles adjacent.

Fig. 3.—Vaccine Vesicle on the Fourteenth Day which has been traumatized.

eralized eruptions. They appear while the local site of the vaccination is open or as late as eight weeks after the primary inoculation, but most often between the ninth and the fourteenth day after inoculation. They have no relation to the size or severity of the local pustule, which may be normal. Among the types of eruptions are the erythematous, urticarial, papular, vesicular, pustular, morbilliform, bullous, pemphigoid, and scarlatiniform. Auto-inoculation by scratching generally occurs an inch or two from the original site, but it may occur elsewhere, as on the eyelid or conjunctiva. The most common type of generalized eruption is undoubtedly the urticarial in its various forms. These include wheals, papules, bullæ, and vesicopapules. The morbilliform are easily differentiated by the absence of fever and coryza and other signs of measles. The scarlatinal forms cause great uneasiness and elevation of temperature. These cases should be observed for urinary complications and subsequent desquamation, in order to exclude scarlet fever. Among the rarer types are the ecthymatous eruptions.

Revaccination.—Vaccination should be repeated after the lapse of ten years, and every five years thereafter. During an epidemic, every one who has not been revaccinated should be vaccinated. Immunity to variola diminishes as we reach the termination of the first decade after the first vaccination. If the revaccination runs a typical course identical with that of the original vaccination, immunity is generally lasting.

OTHER SPECIFIC INFECTIOUS DISEASES.

VI. TYPHOID FEVER.

(*Abdominal Typhus; Ileotyphus.*)

Occurrence.—Of 84 cases of typhoid fever treated by the author, 38 were of the male and 46 of the female sex. The ages were as follows: 1 was of eighteen months, 4 were three years, 9 were four years, 41 were between the fifth and the tenth year, and the remaining cases ranged up to the fourteenth year. Thus 16 per cent. occurred before the fifth year, and fully 50 per cent. from the fifth to the tenth year.

Typhoid Fever and Pregnancy.—According to Etienne, quoted by Morse, the fœtus in utero is born prematurely in 70 per cent. of the cases of typhoid fever in the mother. The causes of the abortion are much the same as those which obtain in pregnant women suffering from any infectious disease. The high temperature, the toxins in the circulation of the mother, and the death of the fœtus, all contribute to cause miscarriage. Of 12 abortions, 9 were stillbirths, 2 lived four and 1 five days..

Fœtal Typhoid.—There are two sets of cases which prove that typhoid fever can be transmitted from the mother to the fœtus: First, those in which the mother, having been infected with typhoid fever, expels a fœtus which may have lived some hours after birth and in whose organs the typhoid bacillus has been found, such as the cases of P. Ernst, Giglio, Lynch, and others. The second set of cases are those in which the blood and fluids of the fœtus give the Widal reaction with bacillosis. Such is the case of Foster and Ballantyne. The mother of this fœtus died from typhoid fever shortly after delivery. The stomach contents and the serum of the peritoneal cavity gave a Widal reaction. The bacillus was found in the kidney, spleen, and intestinal contents, but not in the blood.

Griffith's case was that of an infant apparently healthy, though jaundiced, at birth. When seven weeks old the blood of this infant gave the agglutination reaction. It is possible that in this case the agglutination reaction passed from the mother to the fœtus during the pregnancy without causing typhoid fever in the fœtus. Thus, the presence of the agglutination reaction is no proof of typhoid fever, as it may be transmitted through the placenta, and the fœtus thus escape typhoid fever (Ballantyne).

The anatomical changes found in the fœtus affected by typhoid fever are not identical with those seen in the adult. This is due to the fact that the infection of the fœtus is hæmatogenous, which explains the high fœtal mortality. The spleen is sometimes though not always enlarged. The changes in the gut are not characteristic, being confined to a few enlarged follicles. The liver may be enlarged, and the kidney may show hemorrhages.

Infantile Typhoid.—It has recently been contended that typhoid fever is rare in the infant or the child under two years of age. With the improved methods of laboratory diagnosis of typhoid fever we may shortly be in a position to determine the relative frequency of the disease in the newborn and the young infant. Typhoid fever certainly occurs under the age of two years. As Crozer Griffith has pointed out, we should think of the possibility of its presence in every case of continued remittent fever of the nursling not to be explained on other grounds. Of 331 cases, 9 under two years of age were diagnosed by Henoch as typhoid fever. Among others who report cases are Ollivier, Noyes, Northrup, and Bell. I have seen only 2 cases under two years. Blackader, in a recent series of 100 cases, met 4 under two years of age. Gerhardt reports a case in an infant twenty-five days old, and Blumer 1 in an infant five days old. These cases may be regarded as either congenital or post-natal typhoid.

Morbid Anatomy.—It has been stated that when the fœtus in utero is affected with typhoid fever the process is in the nature of a hæmatogenous infection, and that there are few if any characteristic anatomical changes. In young infants and children the changes in the gut so characteristic of adult cases are not always seen in their full development. The solitary follicles and Peyer's patches are enlarged, but ulcerations are seen only here and there, and seldom lead to perforation (Monti). On the other hand, in older children the changes in the gut closely resemble those of the adult, as has been shown by Henoch. The mesenteric lymph-nodes, especially those in the vicinity of the ileocæcal valve, are enlarged. The remaining changes resemble those seen in the adult subject.

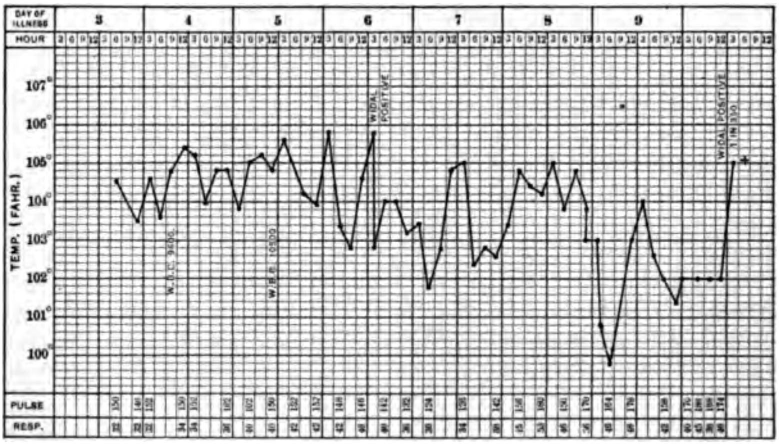
Symptoms.—The invasion of the disease in young children is rarely with a chill. More frequently there are indefinite chilly sensations and mild general malaise. There are headache, pains in the limbs, vertigo, and in many cases vomiting. The symptoms of the period of invasion are so very indefinite in infants and very young children that cases sometimes escape diagnosis.

In other cases, after a few days of malaise the cerebral symptoms become marked. The headache is augmented by delirium at night, especially in older children, and stupor is present. In younger children the period of invasion may simulate a pneumonia. In fact, these cases begin as pneumonia, and it is only on careful consideration of the clinical symptoms—the predominance in a few cases of cerebral symptoms or the enlarged spleen, and the presence of roseola later on, with the elevation of temperature—that we are led to think of typhoid fever. In some of these pneumonic cases there are none of the characteristic features of typhoid. There is no roseola, no splenic enlargement, no epistaxis, but there may be diarrhœa. During an epidemic only the systematic examination of the blood (Widal) will reveal these cases. Such a case is the following: A child, five years of age, was admitted to my hospital service with an indefinite previous history. Temperature 104.6° F. (40.3° C.), pulse 140, and respirations 30. There was apathy, also a bronchopneumonia in the upper lobe of the left lung. This case gave a very positive Widal reaction early in the disease. The spleen became palpable four days after admission. In another case, of a child four years of age, signs of a lobar pneumonia of the upper lobe of the left lung were present without any roseola, enlarged spleen, diarrhœa, or abdominal symptoms. On the fifth day of the disease the Widal reaction became positive in a dilution of 1 : 50. This child died on the sixth day of the disease, with increasing signs of pneumonia and a positive Widal reaction of 1 : 350. Many of these cases of typhoid fever in older children become comatose after the first week. Such a case was recently admitted to my wards. The onset was with headache and fever. There was no vomiting, epistaxis, or chill. The child became unconscious, with a temperature of 106° F. (41.1° C.), rigidity of the muscles of the neck, increased reflexes, ankle-clonus, Kernig's symptom, and enlarged spleen. This case gave a positive reaction to the Widal test, and lumbar puncture failed to reveal anything characteristic in the fluid withdrawn.

The invasion is not characteristic in infants. In exceptional cases (Blackader) a convulsion is the first symptom noted. In some cases there may be a simple continued fever with diarrhœa, without other symptoms. In a case reported by Crozer Griffith the roseola and the enlarged spleen were present.

The subsequent history of a case varies with the character of the infection. In the forms which have a slow, gradual onset the children remain for a time in good physical condition. During the first week the sensorium is clear, the tongue coated, and the face of good color; the spleen may be readily palpable, the roseola appears, and there may be diarrhœa or constipation. In some cases the iliac tenderness is marked; in others absent. It may not be possible to determine the presence of ileocæcal tenderness in young children. The symptoms after the first week may be augmented by delirium at night; in older children this delirium, which has much the same character as in the adult, is also present during the day. Children from five to seven years of age are more likely to have the quiet form of delirium, while older children are noisy and try to get out of bed.

Fig. 40.



Typhoid fever which began as a lobar pneumonia in a girl four years of age. Consolidation of the lower lobe of the left lung; death on the tenth day of the disease.

The course of pneumonic cases is noteworthy. Resolution is tardy in those cases which recover. To the symptoms of pneumonia are added after a time those of typhoid fever—roseola and enlarged spleen. The temperature-curve is not characteristic, and resembles that of the sustained remittent type (Fig. 40). In some cases pleurisy may be present.

In the newly born infant to whom the fever has been conveyed in utero the picture of the disease is unlike that seen in older infants and children. The symptoms resemble those of sepsis of the new-born. Thus in the case published by Blumer the first symptom of the disease was an uncontrollable hemorrhage from the vagina. Before death this was supplemented by hemorrhages into the skin and from the gums.

The cases of typhoid fever in infancy thus far recorded by Morse, Crozer Griffith, Blackader, and the author, may be divided into two classes: those in which there is a mild diarrhœa with distention of the abdomen, roseola, and enlarged spleen; and those which present cerebral symptoms. The latter develop coma, have a distended abdomen, rose spots, and enlarged spleen. In both forms there are severe and mild types. Cases in which the temperature rarely rises above 104° F. (40° C.) recover, while those with a higher temperature may be fatal.

Individual Symptoms.—ROSEOLA.—In children, as in the adult, the roseolar papules are seldom absent. In some cases their number is large, while in others they are few and widely scattered over the surface. They may appear in successive crops, and reappear in a relapse. Occasionally the roseola is preceded by a diffuse erythema closely resembling the scarlet fever eruption. The roseola may, as in the adult, appear on the third, fifth, or tenth day, and may even be delayed until the end of the second week, after which it gradually fades, leaving a pigmented area. The eruption is sometimes so profuse as to resemble the eruption of typhus. It may be profuse in cases in which the cerebral symptoms are very marked. I have seen typhoid fever with severe cerebral symptoms, but with an eruption very sparse or entirely absent at the height of the disease. In severe delirious cases, hemorrhagic areas appear on the bony prominences of the shoulders and extremities. Petechiæ are common. In protracted cases extensive purpuric areas appear on the abdomen. These hemorrhagic cases are not necessarily fatal.

The ENLARGED SPLEEN is one of the most common physical signs. At the outset of the disease it is not always easy to palpate the spleen. This is especially true of younger children. The enlarged spleen is present not only in older children, but also in cases of fœtal typhoid fever. I have seen the enlargement persist for weeks after convalescence. In one case the spleen could be distinctly felt below the border of the ribs for a long time after recovery.

In some forms of relapse the spleen enlarges after having diminished to the normal size. Cases in which the spleen remains enlarged a long time are likely to have slight rises of temperature of short duration. Typical relapses without enlargement of the spleen may occur. The fact that the spleen continues enlarged after the temperature has become normal does not always indicate the approach of a relapse.

Temperature. — An elevation of temperature in young children is usually not observed during the first eight days. Children rarely complain of slight malaise, and a rise of a degree or even more above the normal may escape notice; as a result, the impression is prevalent that the temperature during the first week does not follow the typical curve. The cases which I have observed sufficiently early, and which were not complicated with pneumonia, showed during the first week the gradual rise seen in the adult (Fig. 41). This gradual daily rise of temperature is also seen in relapses. On each day the temperature at its highest point is higher than on the previous day. After the first week the temperature is likely to show a remittent curve with a sustained maximum point. After the second week the temperature may remit, gradually falling, or intermit; frequently it remains high for weeks, with daily remissions. By the end of the second week it reaches 104° to 105° F. (40° to 40.5° C.) at its highest. In the course of the third, fourth, and fifth weeks it may range a degree lower, with remissions to 101° F. (38.3° C.), not reaching the normal. If the case is protracted, the temperature may persist into the sixth week, running up as high as 106° F. (41.1° C.), falling fully five degrees twice daily. In one case the temperature did not become normal until the eighth week. Even at this late period there may be relapses. In many cases the temperature falls to the normal after six or seven weeks, or becomes subnormal, and then after an interval of a few days or a week rises

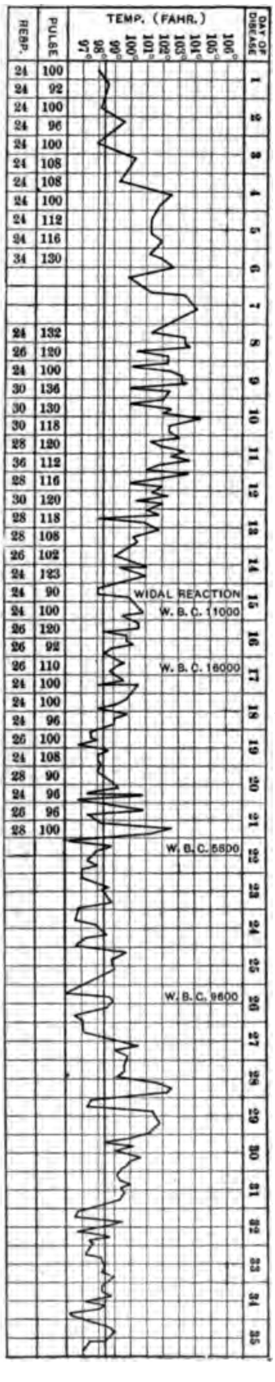


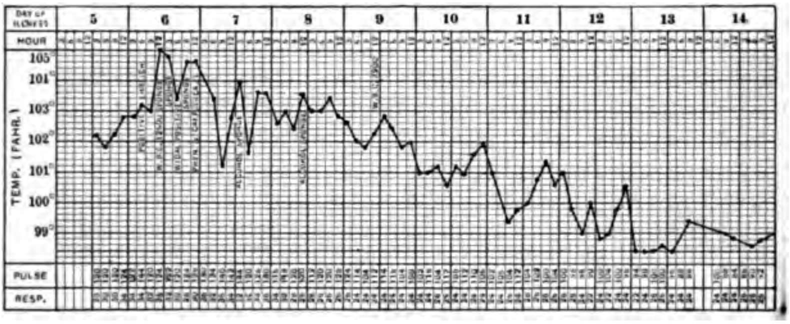
FIG. 41.

Typhoid fever in a boy five years of age, observed from the outset. Widal reaction positive on the fifteenth day.

and fluctuates a degree or more above the normal. This continues for a few days, the temperature remitting to the normal or near the normal. These post-typhoidal fluctuations are sometimes mistaken for relapses. They are rather to be attributed to inanition, or are the result of slight absorption from the gut. In a large number of cases the first sign of convalescence is a subnormal temperature. On the other hand, the temperature may be subnormal for a week or more and relapse follow (Fig. 42).

It may be said that as a rule the first week of typhoid fever in children shows a gradual rise of temperature. The subsequent temperature is sustained, remitting two or more times daily. This curve may last one, two, or more weeks. In other words, there is no characteristic temperature-curve. In relapses the temperature rises gradually from day to day. Among the causes which may give rise to a slight temporary elevation of temperature is constipation. A lobar pneumonia or a bronchopneumonia will cause a persistence of the high temperature, as will also other conditions, such as otitis.

Fig. 42.



Typhoid fever of short duration in a boy six years of age.

The inverted type of temperature-curve is described by Henoch. The morning temperature is higher than the evening, or there may be a rise at 3 A. M. or 6 A. M., a fall in the forenoon, with a rise again at noon, and a fall toward evening. Such a curve may be followed within a day or two by the usual fall in the morning and rise toward evening. These fluctuations occur at the height and at the decline of the disease.

Hemorrhages from the bowel are not so common in children as in the adult. I have seen persistent hemorrhages in only 4 out of 84 cases. In one case I have met post-typhoidal ulcerative colitis. The bowels may be constipated, normal, or diarrhœal. The number of stools varies. In the majority of cases diarrhœa is absent. In some the temperature in convalescence may rise a degree or more for a day or two. In these cases there may be fecal accumulation due to incomplete evacuation of the gut.

Pain.—Sensitiveness in the ileocæcal region is very difficult to determine in young children. In older children it is sometimes marked, and indicates ulcerative processes in that region or in the neighborhood of the appendix.

Pain as a symptom in typhoid fever in the adult occurs in two-fifths of the cases observed by McCrae. In childhood it is not as common a symptom, inasmuch as young children are not apt to complain of pain. It is observed, however, though the exact percentage of cases cannot be stated, on account of the peculiarity of the subjects dealt with. In the adult abdominal pain in the course of typhoid fever is present in complicating pleurisy and pneumonia; or it may be due to a distended bladder, the ingestion of solid food, vomiting, fæcal impaction, diarrhœa, appendicitis, peritonitis, cholecystitis, abscess of the liver, phlebitis of the abdominal veins, and hemorrhage. In childhood some of these conditions may be present, accompanied by abdominal pain. In the cases observed by the author cholecystitis, appendicitis, perforating ulcers, peritonitis, impaction of fæces, and vomiting could be fixed on as a causal factor in the production of the pain. Pain not due to perforation, as a rule, is general in its location. It may be accompanied by meteorism, or may be present with a retracted abdomen. I have seen it in some cases preceded by vomiting; in other cases no such symptom was present. In childhood it is particularly noticeable that pain not due to perforation is unaccompanied by a rise of pulse, and certainly not by a rise of temperature. I have seen very severe abdominal pain, necessitating the administration of opiates, without the least disturbance of the pulse, respiration, or temperature. This latter condition is apt to occur in nervous, hyperæsthetic children. The pain due to perforation will be described elsewhere. I have seen one case where intense pain was caused by a distended gall-bladder with cholecystitis, the diagnosis being confirmed at the operating table. In this case the pain was distinctly localized, and there was temperature due to the hepatic condition.

Otitis is not uncommon. I have seen several cases.

I have observed mastoiditis in 2 cases, 1 of which resulted fatally in the second week of the disease.

I observed **parotitis** in only one case.

The **tongue** of children with typhoid fever resembles that of the adult. It is at first coated, and is protruded in a tremulous manner; subsequently the epithelium is thrown off and the papillæ become prominent. In some cases the tongue resembles the so-called strawberry tongue seen in scarlet fever. At the height of the disease it may become dry and fissured, and sordes may collect on the teeth. The lips become fissured and bleed easily.

The **nervous symptoms** of older children resemble those of the adult. With younger children sopor is the rule and delirium is infrequent. Melancholia or depression occasionally is met with in convalescence, usually in girls of hysterical temperament.

The Heart.—In a recent epidemic of typhoid many cases showed systolic apex-murmurs. These murmurs were loudest over the base, close to the sternum, or over the pulmonary orifice. Such murmurs are myocarditic. In one case there was a loud musical systolic murmur heard over the apex of the heart. It was also heard at the base of the heart. The murmur appeared early in the third week. There was also a pleuropericardial friction-sound. Post-mortem examination revealed myocarditis and pleuropericardial adhesion.

The Lungs.—The occurrence of lobar or lobular pneumonia late in the course of typhoid is serious. At this time the patient's powers of resistance are greatly diminished. Especially grave are the cases which show a sustained high temperature for two or three weeks, and then develop pneumonia. If with the pneumonia there are extensive hemorrhages under the skin at the situation of the bony prominences, the outlook is grave. In such a case I have seen a pneumonia involve the whole lobe of the lung in consolidation within a few hours.

The Blood.—In children, as in the adult, the number of red blood-cells diminishes, and reaches the lowest point at the end of the febrile period. The hæmoglobin also is diminished. The leucocytes are diminished from the outset until convalescence, but increase after it is established. In one of my cases their number fell to 3500, and then rose to 12,400. In a case complicated with extensive ulceration in the gut and bronchopneumonia they numbered 30,000. In fatal cases complicated with lobar pneumonia I have found them as low as 4500. According to Thayer, the polynuclear neutrophiles steadily diminish as convalescence approaches, while the mononuclear lymphocytes and eosinophiles increase. With the establishment of convalescence blood conditions return to the normal.

Relapses.—A relapse is a gradually ascending temperature-curve extending over a week or longer after the temperature has been normal for a time (Fig. 43). A relapse was noted in 7 of 46 cases of my last series. In all, it was mild and no serious results followed. On the other hand, a prolonged low febrile curve causes great emaciation in children. Undue importance has been attached to the condition of the spleen in these cases. The percentage of relapses varies with the nature of the prevailing epidemic. Blackader records 15 relapses in 100 cases, and Henoch 44 in 375 cases. Apparently relapses occur independently of the mode of treatment and diet.

Complications and Sequelæ.—**Skin.**—Subcutaneous abscesses may occur, and onychia is common. Erysipelas and parotitis are rare. Œdema may be confined to the scrotum, or during defervescence the whole surface of the body may be œdematous. In a case of scrotal œdema coming under my observation there were no casts or albumin in the urine; the leucocytes were diminished. Henoch attributes œdema to cardiac weakness rather than to nephritis.

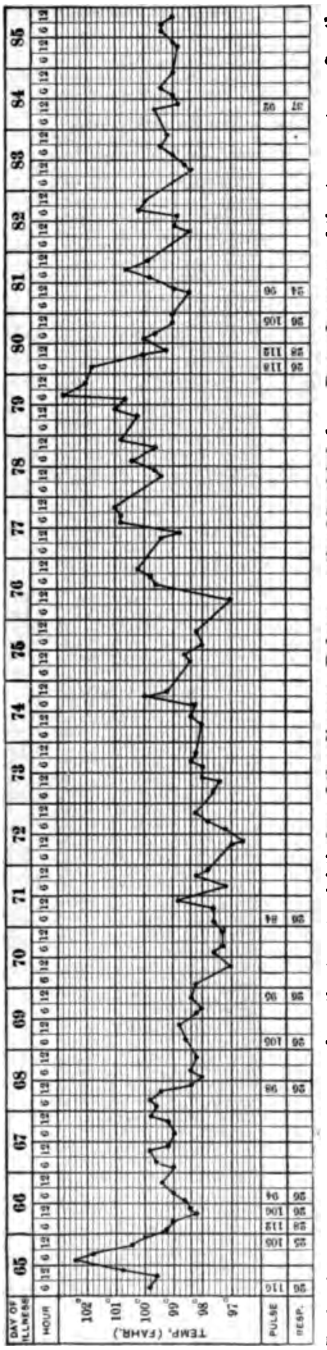
Diphtheria is a very serious complication. I have observed it in 2 of 84 cases.

The Lungs.—Bronchitis is a frequent complication. In the later stages of the disease in younger children it is likely to develop into bronchopneumonia, especially in cases in which the course of the disease is protracted. Pneumonia may occur. Gangrene of the lung is mentioned by Henoch as a rare complication.

Arthritis is uncommon. Usually only one joint is affected. It occurs in the post-typhoidal period and runs a favorable course.

Among the *nervous symptoms* which complicate or follow typhoid fever are aphasia, amblyopia, ataxia of the lower extremities, paralyses of various sets of muscles, double ptosis, and hemiplegia. In hysterical children there may be a post-typhoidal melancholia. In others stupidity may persist for a time. Recovery usually takes place in all forms of paralysis, aphasia, and melancholia. The paralyses are possibly due to a neuritis of toxic origin, as is the case with the other infectious diseases. Hemiplegia occurs only as a result of embolism (Henoch). I have met cases of ataxia and marked melancholia. The children made an excellent recovery. In one case, a boy of four years, catalepsy

FIG. 43.



Typhoid fever: temperature normal on the twenty-eighth day of the disease. Relapse on the fifty-third day. Recrudescence of the temperature after the sixty-sixth day without a true relapse.

was present for a period of five weeks after the temperature had become normal.

Perforation of the Intestine in Typhoid Fever.—The frequency of perforation of the intestine in children affected with typhoid fever, according to all available statistics, is 1.2 per cent. of all the cases. Of my own material of 95 cases there were 3 of perforation, in 2 of which the diagnosis was confirmed by operation (2.1 per cent.). In the adult subject the frequency is 1 to 2.5 per cent. of all cases. Therefore, in the severer forms of typhoid fever in children, perforation of the intestine is almost as frequent as in the adult.

TIME OF PERFORATION.—Most cases of perforation occur in the third week of the disease; a few occur in the second week, and fewer still in the first week of the disease. Perforation may occur in the fourth or fifth week of the disease, or in the relapse.

THE SYMPTOMS OF PERFORATION in typhoid fever are insidious in their onset in some cases, and the disease may have existed for hours before the first symptom noted by most authors as marking the onset of the perforation—viz., pain—is pronounced. In other words, in 2 cases of perforation observed by the author, in which the diagnosis was confirmed by operation, the first symptom supposed to have marked the onset of the disease, six hours before operation, was pain. At this time there was marked peritonitis with fluid in the abdominal cavity, so that perforation must have existed hours before pain was noted. A study of the cases showed that one child (Case I.) complained of sudden vomiting twelve hours before the advent of the pain. Following the vomiting there was a sudden drop in the temperature.

In one of my cases (Case II.) the temperature dropped on the thirty-second day from 102.4° to 97.6° F.; the pulse rose to 160. There was a slight chill, and some blood was passed in the movement, followed by a rise of temperature to 104.6° F.

In the first case mentioned the onset was insidious. The day before marked symptoms set in the child was doing badly. She was somnolent, pale, and took very little nourishment. The pallor increased and vomiting was the first symptom to appear. With the vomiting there was rigidity and tenderness of the abdomen. Twelve hours after the onset the child suddenly complained of pain, which now became the leading symptom. The pain was most marked in the right iliac fossa; the child was nauseated; the rigidity and tenderness of the abdomen became more pronounced, and fluid was diagnosed in the peritoneal cavity. The temperature fell to 100⅘° F., rising again to 105° F. The pulse rose gradually from 126 to 174. The child went into a condition of partial collapse just before operation.

In another (Case III.), that of a boy of twelve, not operated upon, the first symptom appeared on the forty-third day of the disease. The temperature had been normal. From the fourth week on the boy was doing well, when suddenly, on the afternoon of the forty-third day, he complained of pain around the umbilicus; the abdomen became tender and tympanitic, the temperature rose from 100.2° to 104° F., and the pulse rose from 104 to 128.

It can be seen from the description of these cases that in one the onset of the perforation was slow and insidious; in the other two, brusque and quite apparent. In one case the onset was marked by slight vomiting. There was no distention of the abdomen in this case; the pain was slight; the child apparently had a simple dyspeptic attack. The immediate fall of the temperature, however, the change in the facies, the rise of the pulse, the rise of the temperature after the fall, directed our attention to some serious complication. In the second case the drop of the temperature, followed by a chill, with a subsequent rise of temperature, marked the advent of the perforation.

In addition to the symptoms just noted as marking the onset of perforation in typhoid fever, there is an increase in the number of leucocytes. This was true of all the cases mentioned by the author. In one of his cases the leucocytes mounted from 6000 to 7000 to 10,000 to the c.mm., and in another case to 13,000 to the c.mm. The liver dulness in two of the three cases disappeared gradually as the distention of the abdomen and the free gas in the abdominal cavity became more marked. In contrast with the distention of the abdomen in two cases, one presented retraction with rigidity of the abdomen, and in this case the peritonitis was most advanced at the time of operation.

In addition to the symptoms just named, which are characteristic of intestinal perforation in typhoid fever in children, the respirations may become increased in frequency and may be of shallow depth, there is prostration, in some cases collapse, and a distinct change for the worse in the facies. The patients lie prone and resent interference.

It may be stated that when peritonitis becomes more marked the leucocytes, after being increased, may be diminished.

Of the general and local symptoms, therefore, of perforation complicating typhoid fever in children, the most valuable are the initial vomiting, or a chill followed by a drop in temperature, with accompanying abdominal pain, either localized or general, and a subsequent rise of the pulse, rise of the temperature, increase of leucocytes, increase of the local symptoms, such as pain, tenderness, tympanitis, rigidity of the abdomen, with a disappearance of liver dulness. In considering the symptoms seriatim the author is inclined to lay most stress on the sudden appearance of a chill or of vomiting in the course of typhoid fever in a patient who has not hitherto complained of these symptoms, accompanied by slight or marked abdominal pain with rigidity. The fall of temperature is followed by a rise, as hitherto noted; the pain is slight at first, but may be sharp and abrupt in onset, increases subsequently, and reaches its highest intensity within twenty-four hours after the perforation. It is generally localized to the lower part of the abdomen to the right side. It may be paroxysmal in character or constantly present. The distention of the abdomen, though present in most cases, may be absent, and in its place retraction may exist. The liver dulness disappears in most cases according to the amount of abdominal distention.

Prognosis.—The prognosis in intestinal perforation complicating typhoid fever in children varies with the time which has elapsed from the onset of the perforation to the treatment. Fitz has shown that if left alone 5 per cent. of the cases in adults recover. In children we have no corresponding statistics, except that of my 3 cases 1 recovered. This was an undoubted case of perforation in which the inflammation localized itself to the right iliac fossa. Elsberg has included my cases in statistics of 25 cases of typhoidal perforation in children with operative interference, in which the percentage of recovery was 64 per cent., as compared with 22.4 per cent. in the adult. The prognosis, therefore, in children, in mixed statistics, is apparently more favorable than in the adult.

Duration of the Disease.—The duration of typhoid fever varies within wide limits. Henoch, in his tabulation of more than 200 cases, shows that the longest duration was seventy days; the shortest seven to nine days. In my own cases the duration varied widely, if the rises in temperature were taken into account. The average duration in the 95 cases was four weeks and three days. The shortest case lasted ten days, and the longest lasted eleven weeks.

It might be stated in closing these statistics that the average age in my cases was somewhat over eight years. There were 5 cases among them of three years or younger, 1 case being twenty months; another two years of age.

Diagnosis.—Enough has been said to show that the diagnosis of typhoid fever in infancy and childhood is at times very difficult. With young children enteritis, pneumonia, meningitis, and even appendicitis may simulate typhoid fever in their onset. Cases which begin as a pneumonia are especially difficult of diagnosis. The cerebral forms of typhoid fever may closely resemble meningitis. The history is very important. The onset of typhoid fever is gradual, the cerebral symptoms increasing in intensity as the disease progresses. An enlarged spleen and a few roseolar papules will be of service in making a diagnosis, but, on the other hand, an enlarged spleen is common to many conditions of infancy and childhood. In the most puzzling cases, such as those simulating enteritis of non-typhoidal nature, the roseola may at the outset be absent.

In a doubtful case the Widal blood-test should be made daily to clear up the diagnosis. In many cases this reaction is the only clue to the condition. During the prevalence of an epidemic every case of pneumonia or doubtful meningitis or enteritis should be subjected to this test.

The **Widal agglutination reaction** is of greater utility in making a positive diagnosis of typhoid fever in children than in adults. The fact that an enlarged spleen may be due to various causes, such as rickets, the occurrence of fevers of a remittent or continued type, possibly due to otitis, enteritis, pneumonia, and the prevalence of diarrhœa of all kinds in infants and children, tend to make the Widal test of inestimable value.

In a paper based on 84 of my cases of typhoid fever in infants and children, Gershel found the reaction positive in 81. Three hundred and twenty-nine examinations in all were made. Thirty-six were positive on the first test, and forty-five on repeated tests. The reaction appeared in 5 cases on the fifth day, in 3 cases on the sixth day, and in 3 on the seventh day. In other words, 13 per cent. of the tests were positive at the end of the seventh day, 63 per cent. on the fifteenth day, and 89 per cent. on the twenty-fifth day of the disease. The reaction was negative in only 3 cases which gave the clinical symptoms of typhoid fever. These figures correspond to those obtained by Blackader in a smaller number of cases. A negative reaction is of no significance as excluding typhoid fever, whereas a positive reaction is absolutely pathognomonic of the disease. Though 115 of my fever cases were examined by this test for the presence of typhoid fever, exclusive of the above 84 cases, the reaction was obtained in no case in which typhoid was not present. In a few cases the reaction was not obtained until the close of the disease, when the temperature had been normal for some days. In another case of a child of three years, the reaction was not obtained until a relapse had occurred. In the case of a boy of seven years the reaction was not obtained until the third week. It was not taken again until the onset of a relapse in the fifth week, and was negative. It subsequently became positive at the termination of the relapse. This proves that a negative test is of little significance in excluding the possibility of typhoid fever unless the examinations extend over a long period. The presence of the reaction in typhoid fevers which begin with a pneumonia is of interest. In one of these cases, fatal on the eighth day, in a child four years of age, the reaction was negative until the fourth day of the pneumonia. It became positive in an attenuation of 1 : 350 just before the exitus lethalis.

The Ehrlich Diazo Reaction in the Urine.—Thirty-three cases were examined with reference to this reaction. The fifth day was the earliest day on which it was obtained. In the majority of cases the reaction was present from the seventh to the tenth day of the disease. The latest appearance was on the forty-seventh day from the outset of the disease. The reaction was absent in 15 per cent. of the cases. In all of the cases in which the Ehrlich reaction was obtained the Widal test was positive, and appeared in the first two weeks of the disease. The reaction may be present, as it was in one case, on the fifteenth day, and be absent on the next. The diazo reaction may appear before the Widal reaction, but in some cases the contrary is true. In conclusion, it may be said that in the presence of symptoms and signs of typhoid fever the diazo reaction is an aid to diagnosis, although not pathognomonic of the disease.

Of the clinical signs pointing to typhoid fever, the character of fever aids us but little. In the third week it may become intermittent, thus simulating malarial fever. In other cases the fever may be sustained with daily remissions until the fifth week. Typhoid fever with great ileocæcal tenderness and pain may closely simulate appendicitis. In a recent case published by Berg, operated upon for appendicitis, the operation revealed that the patient was suffering from a perforation of the appendix due to an ulcer of typhoidal origin. A continued fever of longer duration than a week, a tremulous tongue, facies, a pulse below 120, an enlarged spleen, and a few roseolar spots, will aid in the diagnosis. The diagnosis of perforation of the gut is not always simple in children. In these subjects tympanites is not uncommon, aside from the presence of peritonitis. I have cited a case in which all signs pointed to perforation, and yet operation revealed nothing. The classical signs of perforation are those of collapse, a sudden fall of the temperature, and a rise of the pulse above its normal frequency, an increase of the leucocytes, and the presence of tympanites increasing until it causes a disappearance of the liver dulness. No one of these symptoms is absolutely pathognomonic. In many cases we shall be compelled to draw conclusions from the general history of the case.

The diagnosis of typhoid fever must, therefore, be confirmed by the Widal reaction, except in a small percentage of cases. The presence of roseola, enlarged spleen, facies, trumulous tongue, diarrhœa, and continued remittent fever are the clinical symptoms which should lead the physician to apply the test.

The **prognosis** of typhoid fever in infancy and childhood is, as a rule, good. The mortality varies with the severity of the infection and the character of the epidemic. If the infection is severe, the complications will militate against recovery. Henoch, in 375 cases had a mortality of 14 per cent.; Blackader, in 100 cases lost only 1; Crozer Griffith had a mortality of 3 per cent.

It is commonly supposed, and some authors lay stress on the fact, that the mortality of typhoid fever in children is lower than in the adult, and therefore the prognosis is better. This simple statement does not give us any idea as to the true mortality of typhoid fever in children. Some authors place the mortality in this disease as low as 4 or 5 per cent. This may be true of some statistics in certain epidemics. In a series of 95 hospital cases of my own of typhoid fever in children, ranging from twenty months to thirteen years, the average mortality, extending over the three years, during which there were three distinct epidemics, was 9.4 per cent. This figure agrees with the percentage of mortality given by Henoch in 350 cases of typhoid fever, and it would about express the average mortality of typhoid fever in children when epidemics of varying severity are taken into account.

In this same material of 95 cases, the mortality in one year was only 4 per cent., and in another as high as 16 per cent. It will be seen from this that hospital cases, from which all statistics are drawn, show that the mortality of typhoid fever in infants and children is much the same as in the adult cases. In my 95 cases there were 9 deaths, distributed as follows:

Below two years of age	2 cases	no deaths
Five years and below	14 "	2 "
From sixth to tenth year	46 "	3 "
From tenth to fifteenth year	33 "	4 "

In this series of 95 cases of typhoid fever there were 15 per cent. of relapses. In this we include only those cases in which there was a true relapse—that is, an average normal temperature for at least eight days preceding the relapse. The average duration of the relapse was eleven days. The mortality in cases where there had been a relapse was nil.

The **treatment** of mild cases of typhoid fever is purely symptomatic. There is little need for the administration of medicines. On the other hand, the severer cases are difficult to manage. This is especially true in the treatment of children, to whom it is not always possible to apply methods adopted with the adult. In cases in which delirium is present night and day bromides in large doses are efficacious. With older children they may prove useless, and morphine may then be necessary to meet the exigencies of the case.

In the vast majority of cases milk forms the basis of the diet. If there is progressive emaciation, one, two, or three raw eggs should be added to the milk daily. It is well in protracted cases not to wait too long for a complete drop of temperature before resorting to other foods than milk. This is especially true of cases extending over a period of seven or eight weeks, in which there is always a rise of temperature of half a degree or a degree above the normal for a few days, with a drop again to the normal or subnormal. In these cases there is a form of inanition fever, post-typhoidal in nature. Solid food should not be withheld too long lest the emaciation become extreme. After the fifth week we may in most cases allow the patient gruels containing cereals. After the temperature has fallen to the normal and remained there for four or five days, it is safe to return gradually to a full diet. It is doubtful if relapses occur as a result of too early feeding if this method is followed. In comatose states resort may be had to forced feeding.

Alcohol is not needed in mild cases. It is given in cases in which the pulse is weak and the temperature high. Delirium is no contraindication to its use, as it is in other affections.

The **heart** is stimulated by digitalis, strychnine, or camphor. If the heart has shown slight dilatation with a murmur developing in the course of the disease, the patient should not be allowed out of bed too soon for fear that unfavorable symptoms may result.

The temperature is controlled by hydrotherapy. The patient is placed in a bath at 100° F. (37.7° C.), and the temperature of the water gradually reduced to 85° F. (29.4° C.). With older children the temperature may be lowered still further. Children do not bear the classical Brand bath treatment well. The plunge bath is given three or four times daily whenever the temperature is 103° F. (39.4° C.) or more. Should the child struggle very much against the administration of the bath, it is wiser to forego it and substitute sponging. If the sponging is not followed by good reaction, the use of water should be abandoned. In exceptional cases of delirium a bath once or twice daily at 105° F. (40.5° C.) has a quieting effect.

Hemorrhages from the bowel are not frequent in children. They may occur early or late in the disease. In the latter case they must be differentiated from hemorrhage due to enterocolitis of a post-typhoidal character. In hemorrhage due to typhoidal ulcer an ice-bag is applied to the abdomen, and small doses of opium, preferably the deodorized tincture, are administered to control peristalsis. Ergot and digitalis are given internally in order to contract the bloodvessels if possible. Enemata should not be given. If the hemorrhage becomes excessive, it is proper to give hot saline enemata, and to infuse normal saline solution under the skin or into the veins.

Enteritis of an ulcerative or pseudomembranous character occurring as a complication of typhoid fever is treated in the same manner as the primary affection.

Perforation should be treated on surgical principles. As with adults, those perforations which occur late in the disease, when the patient is in an exhausted and emaciated condition, give a less favorable prognosis than those which occur early. The surgical treatment will be more successful the sooner the diagnosis is established, for in those cases in which peritonitis has advanced to a marked degree the prognosis is fatal. The success of surgical treatment will also depend largely on the fact as to whether the perforation is single or multiple. In one of my cases it was demonstrated at operation that no less than three ulcers had perforated, and there were as many more on the point of perforation, so that in this case simple sewing up of the ulcerated parts could scarcely have succeeded in saving the patient, for in this very case a perforation after operation caused the death of the patient. In such cases the treatment of multiple perforations is a problem for the surgeon.

Constipation.—In most cases of typhoid fever an enema will remove accumulated feces from the lower bowel. Enemata are not given unless indicated. If the bowel contents are streaked with blood, enemata should be discontinued. In cases in which there is a slight rise of temperature during convalescence without apparent cause, grains v (0.3) hydrarg. cum creta should be given. Tympanites is treated as in the adult subject. The evacuations should be mixed with an equal volume of a solution of carbolic acid (1 : 20) as soon as passed. The hands of the nurse should be thoroughly cleansed after each movement. The patient's hands are cleansed daily, in order to avoid auto-infection.

VII. MALARIAL FEVER.

(*Paludism; Malaria; Intermittent Fever.*)

Malarial fever is an acute infectious disease due to the inoculation of the individual with the Plasmodium malariæ. It is common in infants and young children, and is believed to occur in utero. Crandall has reported a case in which symptoms developed eighteen hours after birth, and in which the plasmodium was found in the blood of the infant. Those who, like Moncorvo of Brazil, have opportunities to observe malarial fever in young infants and children, find the greatest frequency under two years. The author has not met paludism as frequently in the nursing infant as in older children. The reason for this must lie in the fact that young infants are more protected from infection with veils, etc., than older children. One attack does not confer immunity to subsequent attacks; on the contrary, infants and children once the subject of paludal poisoning seem particularly liable to reinfection and relapses.

The period of incubation varies from a few hours to weeks. In the tertian type it is believed to be from seven to fourteen days. In one of my cases the first chill appeared eleven days after the patient had left the malarious district.

Etiology.—The essential cause of malarial fever is the same in infants and children as in the adult. It is an inoculation fever, and is conveyed to the human subject by a certain species of mosquito (Anopheles). The poison exists in the neighborhood of swamps and stagnant waters.

The Parasite.—The plasmodium or protozoa of malaria circulates in the blood of infants and children, undergoing its cycle and sporulation in the same manner as in the adult. In one series of cases in infants and children that I studied, the tertian was the most prevalent form of parasite. These cases occurred in New York City and its vicinity. This has been the experience of other New York City observers. One may assume that the blood will, as a rule, contain the parasite prevalent in a given locality. Several forms of parasites may exist in the blood of the same child, or there may be several generations of the same plasmodium. These may mature at different times, giving various types of fever in the same subject. In a tertian case, the fever may thus become quotidian, a second set of parasites causing a distinct chill and fever (paroxysm) on the day when the first generation is quiescent. We may have, as Mannaberg and others pointed out, simple and double tertians and quartans. But no combination of quartan parasites can simulate the simple tertian type. I have seen very few cases of quartan in children. They are uncommon in New York City, but I have seen preparations of the quartan type which were found in the blood of children in the Southern States. As in adults, tertian paroxysms may occur every day, caused by two sets of parasites which mature at about the same time daily, or one set matures at a different hour than the set of the following day. In such a case paroxysms would occur at the same hour only every other day. Many children have a distinct severe paroxysm only every other day, but on the intervening day a careful examination will detect a very low fever. This is probably due to a set of parasites which mature without producing marked chill or fever (abortive).

The Blood.—In recent tertian I have found young spores in abundance in the blood a few hours after the chill. In some specimens the spores were free. Between paroxysms in tertian cases the blood contains colorless oval plasmodia—the fully developed body—leucocytes having rods and pigment-granules and rarely, small round forms with flagellæ (Koplik). In stained specimens (methyl-blue) young native forms are found in all stages up to fully developed protozoa. The red blood-cell containing the parasite is distinctly enlarged. I have found in the stained specimen as in the unstained ones, the sporula in free groups, bodies with flagellæ, and erythrocytes with stained granules. The half-moons are also found in chronic cases. The blood contains free granules, and peculiar shrunken, brassy-colored, red blood-cells. Monti found the specific gravity of the blood to be increased.

Morbid Anatomy.—Post-mortem examinations in cases of malarial fever in infants and children are exceedingly rare. Opportunity may be afforded when death occurs as the result of accident or of some other disease. Monti states that in fatal cases the spleen is enlarged; the capsule is tense, and in places shows rupture. The pulp is dark red owing to pigment deposit (melanin). Old spleens show a disappearance of melanin and a deposit of yellow ochre pigment along the trabeculæ. In chronic cases the connective tissue is increased, the liver is enlarged, and there is atrophy of the liver-cells. The parasites are found in the blood. The endothelium of the bloodvessels contains yellow and brown pigment. In exceptional cases there are melanin deposits. In acute cases the bone-marrow is the seat of melanin deposit; later this disappears, and the marrow is found to be yellow and fatty. The brain cortex in severe cases shows pigment deposit; sometimes there are thromboses and hemorrhages.

Symptoms.—Children living in malarious districts do not always manifest malarial poisoning by having paroxysms of chills and fever. The disease is masked under the form of a progressive anæmia, with accompanying enlargement of the spleen. These patients develop symptoms in from a few days to a few weeks after leaving the malarious region. The period of incubation is thus distinctly indicated.

The onset of a paroxysm is usually marked by the appearance of chills. In young infants a distinct chill is not always present. They become cold and blue at a certain time each day. In older children the paroxysm is indicated by headache and a feeling of lassitude, which comes on at a certain time each day, or by a distinct chill. In exceptional cases eclampsia or vomiting may usher in a paroxysm. In other cases there is no eclampsia, but the hands become cold, there is a feeling of faintness, and the child complains of being ill. Meanwhile there is a rise of temperature, during which there are muscular tremors of the extremities and a peculiar upward rolling of the eyes, indicating an impending convulsive seizure. The chill may occur during sleep. In one case the mother noticed that the child (three years of age) became pale during sleep, the hands and extremities became cool, and the pulse rapid. The febrile movement following the chill may be very slight, scarcely half a degree above the normal. In such cases the chill is not marked or is scarcely noticeable. This occurs in double tertian, in which one paroxysm is abortive. In most cases the fever is very high at first—so high that it is characteristic. A temperature of 106.5° F. (41.3° C.) is not uncommon, and is well borne. As a rule, the fever has a distinctly intermittent type. The temperature may rise after the initial chill and remain high for days, and then fall to the normal. In the simple form the fever lasts from four to twelve hours, and is followed by a critical perspiration, during which the temperature rapidly falls to the normal. In some cases the children appear free from symptoms in the interval between the paroxysms. Others suffer from headaches and a feeling of lassitude, and in infants there are gastric and intestinal disturbances. In protracted cases a distinct anæmia develops, with progressive enlargement of the spleen. Neuralgia of the peripheral nerves has been noted in older children.

During a paroxysm Monti noted polyuria, which persisted until the following day.

The spleen enlarges rapidly, and in a short time may be felt as low down as the umbilicus. I have found the spleen markedly enlarged; in one case the organ was not palpable below the ribs, although a slight enlargement could be detected on percussion.

The liver may be enlarged in chronic cases.

In subacute forms chills are not present, but there is an irregular febrile movement, with progressive anæmia and splenic enlargement.

Repeated Attacks or Relapses.—Children, as well as adults, may have repeated attacks of malarial fever. As a rule, however, these so-called independent attacks in children are relapses, due either to inefficient treatment or to the development of a new series of parasites. Infants may have relapses. I have treated such cases until all anæmia and signs of active malarial poisoning had disappeared, and then administered arsenic for months, only to find a return of the symptoms after an interval of months.

The **diagnosis** of malarial fever is based upon an examination of the blood. If a child suffers from pronounced anæmia, malaise, pains in the limbs, and enlarged spleen the blood should be carefully examined. Expert knowledge is always necessary for a definite diagnosis. It is surprising to note the large number of cases beginning with chills and presenting an intermittent fever curve and enlarged spleen, diagnosed as malarious, in which parasites cannot be detected in the blood. Many septic and inflammatory processes in infants and children simulate malaria. Rachitis, syphilis, gastro-enteric catarrh, otitis, pneumonia, typhoid fever with relapses, have all been mistaken for malarial fever. The diagnosis rests on an examination of the blood in all cases in which chills and fever or any of the symptoms described coexist with enlargement of the spleen.

Quinine should not be administered until the blood has been very carefully examined. In other words, malaria should be diagnosed or excluded before resorting to this remedy, which was formerly much in vogue as a diagnostic test. Its use before diagnosis can only result in uncertainty, since there are rises in temperature, not due to the paludism, which may be influenced by quinine. A very high temperature of an intermittent type, in connection with other physical signs, should cause the physician to consider the possibility of paludal poisoning.

I have not seen cases of the pernicious type. They occur in the Southern States.

Acker has recently published 2 cases of malarial fever in children, in which there were the initial cerebral symptoms of coma and convulsions. Coma in one case came on in paroxysms. In the interval the child was rational. The æstivo-autumnal parasite (pernicious) was found in the blood.

The **prognosis** of malarial fever in New York City is very good. With proper treatment the patient should recover. I have never met a fatal case. They occur in districts in which the pernicious type of the disease is prevalent.

Treatment.—If possible, the patient should be removed from the malarious district. The remedies employed in all cases are quinine and arsenic, or their derivatives.

According to Golgi, quinine should be given before the paroxysm, and also in the intervals. The action of the drug is exerted directly upon the plasmodium. At this time segmentation of the parasite takes place in the blood, and most of the young parasites are free in the plasma. They then respond most quickly to quinine. Large doses should be given to infants and children, in order that the infection may be destroyed quickly and completely. The soluble bisulphate and muriate are suitable preparations. To an infant under one year of age grains ij (0.1) are given in a dose, repeated three times a day, the last dose being given from three to five hours before a paroxysm. To children between two and five years of age grains iij to v (0.2 to 0.3) are given in the same manner. Some infants take quinine readily when it is suspended in powder form in milk or water; others are given a piece of chocolate, and when the surface of the mouth is coated with the candy the drug is administered. Euquinine is a preparation tasteless and ordorless, and is readily taken by children. The dose is the same as that of quinine. The syrup of yerba santa is also a good menstruum. In cases in which children cannot take quinine by mouth, Jacobi advises giving it per rectum, dissolving the drug in a solution of tartaric acid. I have never been forced to use subcutaneous injections of quinine, as the pernicious form of malaria in which this mode of therapy is principally resorted to is not prevalent in New York City.

Infants and children with chronic or subacute forms of malaria are likely to be constipated. Under these conditions I have found calomel more efficient in clearing the gut than castor oil.

After the quinine treatment has been continued for some time the spleen will be observed to diminish in size and the paroxysms to disappear. If the anæmia persists, it is well, after diminishing the frequency of the dosage of quinine, to combine it with small doses of Fowler's solution. The arsenic must occasionally be temporarily discontinued, or the functions of the stomach will become deranged. Warburg's tincture does not seem to be very efficacious with children under five years of age, nor with older children, unless given in very large doses. Children do not develop cinchonism as quickly as adults, and the quinine may therefore be continued for a long time. Treatment should not be suspended until the spleen is no longer palpable and the anæmia has disappeared. Quinine should then be continued in small doses at regular intervals.

The preparations of cinchona, such as cinchonidia, cinchonidin, chinidin, etc., are not reliable. The following is Baccelli's formula for the subcutaneous use of quinine in pernicious intermittent fever:

Quinin. muriat.	15 grs.	(1.0).
Natrium chlorat.	1 gr.	(0.06).
Aq. destillat.	ʒiiss	(10.0).

VIII. INFLUENZA.

(*La Grippe; Acute Catarrhal Fever.*)

Influenza is a specific infectious disease chiefly affecting the mucous membranes. It is highly contagious, although all individuals exposed do not contract the disease. It occurs in the form of pandemics in which whole communities are affected. This pandemic form occurs less frequently in children than adults, and is of interest to the physician only when an epidemic prevails. The endemic form of influenza affects children more frequently than adults, and is the form which will be described, although in its symptoms it closely resembles the epidemic form. The endemic form may occur at any season of the year. In large cities influenza is always present (endemic), and appears to be more prevalent after rapid changes from lower to higher temperatures. Rapid fluctuations in the humidity of the atmosphere in winter also favor the development of the germs of this disease. In New York City, midwinter and spring are the seasons when outbreaks of this affection occur.

Age.—Influenza may affect the newly born infant. A case of this kind is reported by Townsend in the *Transactions of the American Pediatric Society.* The disease is most frequent between the ages of six months and five years. The younger the child, the more severe the affection.

Mode of Infection.—Individuals are infected by coming into contact with others suffering with the disease. The germ is contained in the sputum and the nasal secretions; therefore poorly ventilated rooms and public conveyances favor the transmission of the disease. Parents may transmit it to their children in the act of kissing, and wet-nurses who have la grippe are likely to infect the infant at the breast.

Etiology.—The epidemic form of influenza has been studied by Pfeiffer and Kitasato. Pfeiffer isolated a bacillus from the bronchial mucous membrane, trachea, and lungs. This bacillus, which is now believed to be the essential cause of epidemic influenza, is exceedingly small, and two or three times as long as it is broad. It has rounded extremities, occurs in pairs and chains, does not stain by Gram's method, and in influenza, pneumonia, and encephalitis is found in enormous numbers in the lungs. It is called the Bacillus influenzæ. It is still an open question whether it occurs in the blood. Although this bacillus has been found in sporadic cases of endemic influenza, competent observers, Luzzato among the latest, have found that in a large number of endemic cases of influenza the Pfeiffer bacillus is absent. In its place is found the Fränkel diplococcus. This is thought to be the essential cause of an important group of cases of endemic and sporadic influenza in children—the so-called pneumococcus grippe. Predisposing elements in the etiology of endemic influenza are exposure to cold and a diminution of the strength of the individual. One attack does not protect the individual from subsequent attacks.

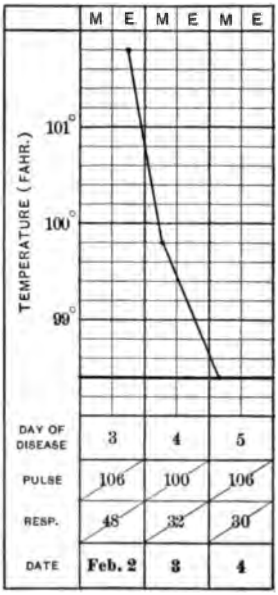
Incubation.—Influenza is believed to have an incubation period of from twelve hours to three days. Endemic influenza occurs frequently in large cities and at times local epidemics of the disease are seen.

Morbid Anatomy.—Inasmuch as influenza is rarely fatal, the pathological anatomy is imperfectly formulated. In fatal cases a general inflammatory condition of the mucous membrane of the nasal passages, and of the larynx and trachea, is found. The surface of the lining membrane of the bronchi is reddened, covered with mucopus, and the membrane itself is infiltrated with small round cells. There may be a diffuse inflammation of the smaller bronchi, with peribronchitis and inflammatory reaction. Areas of bronchopneumonia or lobar pneumonia are found in the lungs. The heart is dilated and the seat of myocarditis. There may be endocarditis and the kidneys may present an acute nephritis. The pleuræ are inflamed, and there may be serous or serofibrinous pleurisy or empyema.

Among the other lesions are those due to the complications, otitis, meningitis, inflammation of the gastro-intestinal tract, and cerebrospinal meningitis.

Symptoms.—It has been customary to divide the symptomatology of endemic influenza as it occurs in children into clinical forms. According to my experience, there is no sharp dividing-line between the various forms of endemic influenza as seen in children. The gastro-intestinal, nervous, and pneumonic forms are frequently present in the same patient. Endemic grippe as it occurs in children in New York City will be described, the epidemic or pandemic form being ignored.

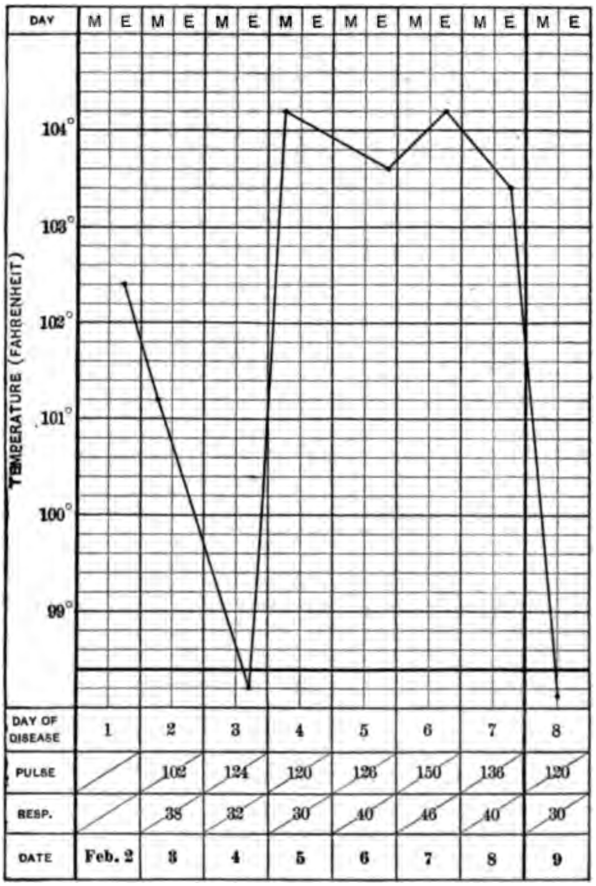
FIG. 44.



Endemic influenza with bronchitis in an infant seven months of age.

The most frequent form is the catarrhal of an acute and even subacute type. The infant or child may at the outset have a chill. Most frequently there is vomiting, and also fever, and pains in the head and limbs. There is a coryza, and in many cases a croupy, barking cough. The eyes are injected, the face is red and flushed, and the child presents an appearance resembling that of the first stage of measles. The mucous membrane of the throat is deeply injected and the tonsils inflamed and enlarged. The temperature is elevated; in fact, at the outset it is as high in this disease as in malarial fever, 106.5° F. (41.3° C.). The cough is sometimes incessant. The irritation in the throat is extreme, and vomiting after the coughing paroxysm may lead the physician to believe that he is dealing with whooping-cough. In young infants these symptoms may last for a day or two, during which the movements may become green and even diarrhœal. This diarrhœa is sometimes so severe as to be a prominent feature of the disease. The prostration both in infants and children is marked. After two or three days the catarrhal condition of the upper air-passages subsides, and the patient develops symptoms of an acute bronchitis of a severe type. These forms of grippal bronchitis have at the outset a high febrile curve (Fig. 44), and a fever persisting for days. The bronchitis affects the smallest bronchi. There may develop a bronchopneumonia in small areas. In other cases the bronchitis passes suddenly into a pneumonia without a preceding chill. The pneumonia of la grippe may be lobular or lobar in type. In the vast majority of cases the pneumonia is of the pneumococcus variety. Especially severe are the cases of grippe which are ushered in with a chill, high fever, and cerebral symptoms, such as sopor, delirium, and rigidity of the neck muscles. In many of these cases examination of the chest reveals pneumonia. These cases are not so common among infants as among older children. Cases in which there is a cerebrospinal infection in no way differ from cases of cerebrospinal meningitis due to the meningococcus or the pneumococcus. The endemic grippal forms of cerebrospinal meningitis may be caused by the influenza bacillus (Sänger) or the pneumococcus. The child at first complains of fatigue, and has a tendency to sleepiness, cries out and starts in its sleep, and suffers from intense headache. After a time vomiting with rigidity of the muscles of the neck sets in. These symptoms increase in intensity, sopor finally setting in with all the symptoms of a cerebrospinal meningitis. These cerebral cases are rare. A common form of grippal attack is that in which all the symptoms of nasopharyngeal inflammation are present. There is also mild bronchitis of the larger tubes. The temperature may fall to the

FIG. 45.



DAY OF DISEASE	1	2	3	4	5	6	7	8
PULSE		102	124	120	126	150	136	120
RESP.		38	32	30	40	46	40	30
DATE	Feb. 2	3	4	5	6	7	8	9

Endemic influenza, lobar pneumonia of the lower lobe of the right lung. Child two and one-half years of age.

normal in the morning or toward noon, but toward evening it rises from one-half a degree to three degrees above the normal (Fig. 43). The child plays in the afebrile intervals. It may awake from sleep in a peevish, irritable mood, or may start in its sleep. These symptoms may continue for a week or longer. In many of these cases there is serous or purulent otitis media, or there may even be a mastoid inflammation from the outset. In other cases the patient has an intermittent or remittent fever. The fever, if a continued one, has morning or evening remissions. Examination of the heart may reveal an acute endocarditis, although marked symptoms of cardiac involvement may be absent.

Symptoms referable to the kidney have received little attention in text-books. In endemic grippe there is almost always a slight trace of albumin in the urine, which, as a rule, disappears at convalescence. Occasionally, there is a true nephritis, with casts, decreased secretion, and blood. Such cases have been described by Freeman. Of grave import are the cases of nephritis in endemic grippe which at first show a trace of albumin and a few hyaline, epithelial, and blood-casts, with a very small (microscopic) amount of blood in the urine. The urine is normal in amount. The condition is revealed only by the microscope. Œdema is absent. The child is at first pale, but this pallor disappears later. The trace of albumin in the urine, however, with a few casts and blood-cells, persists for months. These cases have been described as "cyclic" albuminuria. They are really nephritis of an insidious character following endemic grippe.

I have seen cases of endemic grippe complicated with swelling of the parotid and submaxillary glands and of the lymph-nodes of the neck.

The **duration** of endemic grippe is from two or three days to as many weeks. I have seen cases present a temperature-curve for three weeks, but have not met the cases of protracted duration, with or without fever, described by Filatow, and would regard such cases as peculiar to the country of that author.

The **prognosis** of endemic grippe is favorable. If complications supervene, it varies with their nature.

The **diagnosis** presents no difficulties. In some cases the nervous symptoms may cause the physician to suspect meningitis when pneumonia is present. A careful physical examination will dispel the doubt. Meningitis and pneumonia may be present in the same case. Otitis may supervene without the presence of marked symptoms referable to the ear. An aural examination by an expert should be made in all cases in which fever persists and physical examination of the lungs and other organs fails to reveal abnormal conditions.

The **treatment** of la grippe is simple. At the outset in the milder cases small doses of quinine are administered, to control the headache, restlessness, and fever. For the angina small doses of ferric chloride are given to infants every one to three hours. In older children, the throat is, in addition, sprayed two or three times daily with salt solution or a solution of boric acid or listerine. The fever is treated by sponging; packing or baths are rarely necessary. The bowels of infants are washed out with high enemata if diarrhœa sets in, and milk food is temporarily suspended. Pneumonia, if present, is treated as outlined in the section on that disease. Otitis should be treated by early incision of the drum-membrane, as even cases in which no pus, but only serum, is present are relieved by this procedure. With older children the use of phenacetin alone or in combination with monobromate of camphor is permissible if the headache and pains in the limbs are very troublesome. A grain of each may be given once or twice daily for a short time. The prostration is best combated by the use of strychnine alone or combined with caffeine. Whiskey is not well borne in these cases, since it is likely to cause gastro-intestinal symptoms.

IX. GLANDULAR FEVER.

(*Pfeiffer.*)

Glandular fever is a form of infection which manifests itself by an enlargement of the lymph-nodes of the neck, with accompanying enlargement of the liver and spleen, and an initial period of fever. It occurs from the second to the eighth year of life. During an extensive epidemic J. P. West observed it in the nursing infant.

The **etiology** is obscure. This disease is a species of infection or toxæmia. In some cases (West) there has been diarrhœa, in others constipation, and in most cases a slight injection of the nasopharynx. It is possible that the infectious agent gains access to the lymph-channels through the gut or nasopharynx. This would account for the involvement of the mesenteric glands, as observed by Pfeiffer, and for the infection of the nodes of the neck through the thoracic duct.

Symptoms.—After slight malaise, or even without prodromata, children are attacked with fever, restlessness, headache, vomiting, and pains in the limbs. After a few hours of these premonitory symptoms, swelling of the cervical glands on one or both sides is noticed. These glandular swellings extend from beneath the body of the jaw along and beneath the upper third of the sterno-mastoid muscle. The lymph-nodes beneath the muscle are also affected. After one or two days these glands or nodes not only increase in size, but nodes at the back of the neck and in the supra-clavicular region are also affected. In the cases recorded by West the axillary and inguinal lymph-nodes were also involved. The temperature at first ranges from 102° to 104° F. (38.8° to 40° C.), but in from twenty-four to forty-eight hours it may fall by crisis. There is a slight redness of the pharynx or the color of the mucous membrane may be normal. There is pain on deglutition, and there may be a slight cough, but no distinct pulmonary affection. In both Pfeiffer's and West's cases the liver and spleen were enlarged. In the cases of Starck, Rauchfuss, and Protussow these enlargements were not always present.

The **lymph-nodes** may enlarge to the size of a pigeon's egg. The redness of the pharynx is disproportionate to the enlargement of the nodes (Rauchfuss), so that it is hardly permissible to speak of an anginal lymphadenitis, as in scarlet fever. In both Starck's and West's cases there was enlargement of the nodes, which were not painful, but sensitive to pressure. The swelling of the carotid lymph-nodes began, as a rule, after a few hours, was in most cases first visible on the left side of the neck, and reached its height from the second to the fourth day. The glands on the opposite side of the neck then became affected. The swelling rarely continues unilateral. It is uniform, as thick as an index-finger (West), and is composed of several nodes. There is a stiffness of the neck and also a sensation of choking. Suppuration is absent. There is in all cases a tenderness of the abdomen about the umbilicus, which, in Pfeiffer's opinion, indicates an infection of the mesenteric nodes. West found the mesenteric nodes enlarged in 37 cases.

Diagnosis.—The disease is readily differentiated from mumps. In some epidemics the submaxillary glands were involved, but never the parotid. The appearance of the swelling of the lymph-nodes first on one side, and then on the other side of the neck is characteristic, and should be differentiated from the glandular swellings occurring with grippal affections or pneumonia. Heubner has reported cases in which there was a complicating nephritis.

Duration.—The fever disappears after a few hours or may last two or three days. It may recur later. The gandular swellings, however, increase or persist nine to twenty-seven days, the average duration being sixteen days (West, Rauchfuss).

Treatment.—As the affection has a tendency to spontaneous recovery, the treatment is purely symptomatic.

X. MENINGITIS.

Classification of the Different Forms of Meningitis.—The simplest classification is that which divides meningitis into the primary and secondary forms. The primary form includes cerebrospinal meningitis of the epidemic type, or cerebrospinal fever, as also the sporadic forms of this disease, and, as a separate entity, the pneumococcus meningitis. In the secondary forms we have the tuberculous pneumococcus meningitis, the latter being secondary to pneumonia, endocarditis, or injury of the cranial bones. Third, there are the pyogenic forms of meningitis, due to staphylococci, streptococci, or secondary either to the disease of the cranium or local infections. Fourth, there are the forms of meningitis secondary to typhoid fever, influenza, diphtheria, gonorrhœa, syphilis, anthrax, actinomycosis. Fifth, in a separate rubric there is the so-called serous meningitis, which is recognized as a secondary form of the disease, due probably to streptococci or pyogenic organisms. It will be seen that this classification recognizes both the sporadic and the epidemic forms of the cerebrospinal fever as the same disease due to the same essential cause.

Barlow and Gee divide simple meningitis in infants and children, as to locality, first, into the vertical form, which is a leptomeningitis, and affects the vertex of the cerebrum, sometimes spreading toward the base, and often involving the cord; and in the second class they include the so-called postero-basic forms of meningitis, in which the exudate is confined principally to the posterior part of the base of the brain.

In constructing this section the author has utilized 110 cases of meningitis occurring in his hospital service. They were divided into the following groups: 68 were cases of the cerebrospinal form of meningitis of the epidemic type. Of the remaining cases, 35 were tuberculous forms of meningitis, 1 case a so-called staphylococcus meningitis, 1 case a primary pneumococcus meningitis, 3 cases streptococcus meningitis, and in 2 cases a bacillus was found resembling the influenza bacillus in cultural characteristics.

The author will first consider cerebrospinal meningitis of the epidemic and sporadic type, and then will consider the so-called vertical meningitis and postero-basic meningitis of Barlow and Gee, serous meningitis, and finally tuberculous meningitis.

Cerebrospinal Meningitis.

(*Cerebrospinal Fever; Spotted Fever; Petechial Fever; Malignant Purpuric Fever.*)

Cerebrospinal meningitis is an acute infectious disease, the characteristic lesion of which is an exudative inflammation of the pia mater of the brain and spinal cord. It occurs in epidemics, but may occur sporadically.

Etiology.—Cerebrospinal meningitis, both in its epidemic and sporadic forms, is due to an infection by the Diplococcus meningitis intracellularis of Leichtenstern, Weichselbaum, and Jäger. This micro-organism is a diplococcus reminding one strongly in its form of the gonococcus. It is decolorized by the Gram stain. It is found not only in the body of the pus-cell—hence its name—but in the exudate also outside of the pus-cell.

Though the epidemic form of cerebrospinal meningitis is caused in the vast majority of cases by this micro-organism, there is another group of cases of the cerebrospinal type which is caused by the Diplococcus pneumoniæ. This latter class of cases has been described by Netter, Foa, and Bordoni-Uffreduzzi. These cases may occur epidemically also, but are generally seen in combination with lobar or broncho pneumonia, or as a complication of otitis media. The form of affection discussed in this section is rather the sporadic and epidemic type of cerebrospinal meningitis caused by the intracellular diplococcus above mentioned. In the epidemics of this disease so far observed, it is not unusual for several members of a family to be attacked. The rule, however, is the contrary. The cases in an epidemic number several hundreds, the last epidemic in New York amounting to somewhat over 1000 cases.

The disease seems to have no marked tendency to spread. In large cities the epidemics occur in the spring of the year; and, after the epidemic has run its course, sporadic cases are observed in the fall and winter months.

Mode of Infection.—It has been a matter of great speculation as to how the infection is conveyed from person to person in this disease, if such does occur; and also as to the manner in which the micro-organism—the intracellular diplococcus—gains access to the circulation. Cases are observed here and there, and I have seen two such cases in the last epidemic, in which the disease is complicated by pneumonia, the meningitis and the pneumonia both being due to the intracellular diplococcus. These cases, however, are exceptional. It has been supposed that the micro-organism gains access to the circulation through lymph-spaces in the mucous membrane of the nose and conjuctivæ.

I have published one case in which the Diplococcus intracellularis was found in the secretion of the conjunctiva in a child suffering with the disease, in whom the meningitis had been preceded by a conjunctivitis. Wright has published a case in which the intracellular diplococcus was found in the nasal secretions of a person suffering from influenza symptoms, mild headache, fever, and constitutional disturbances, which might very well have been a mild form of cerebrospinal meningitis. A micrococcus, so-called Micrococcus catarrhalis, is found in the normal secretions of the nose, and it has been mistaken time and again for the Diplococcus intracellularis.

It has been intimated that the infection may gain access to the circulation through the respiratory organs. However these facts may be, they do not definitely establish how the infectious material gains access to the circulation, or whether the disease is conveyed from person to person.

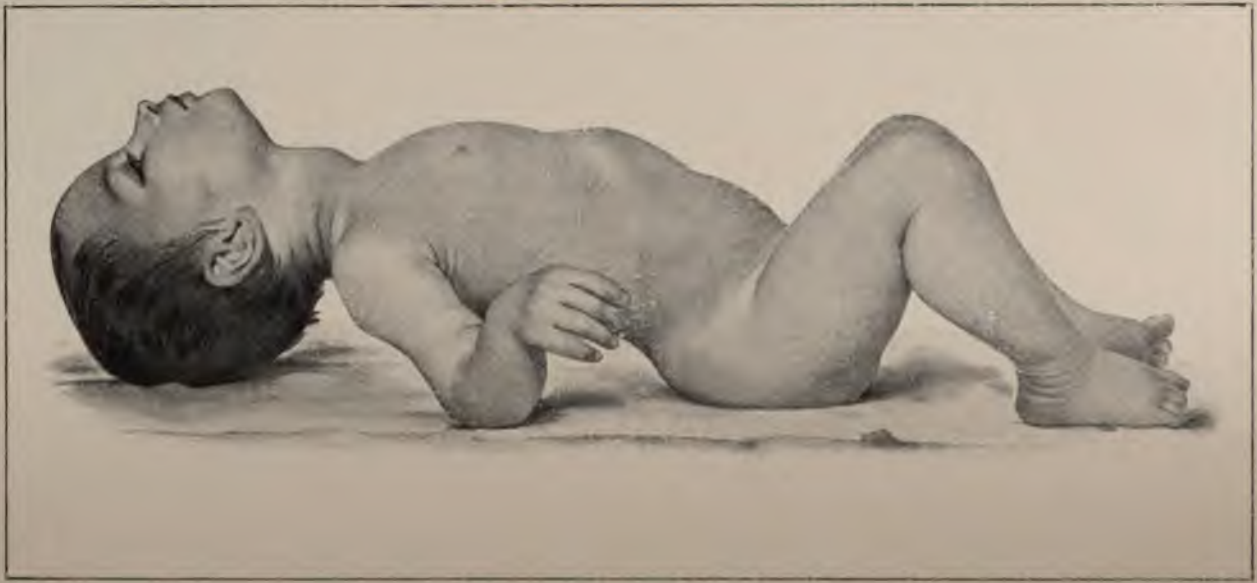
Occurrence.—Cerebrospinal meningitis is distinctly a disease of young people. Rotch reports a case in an infant six days old. The youngest case of the epidemic type seen by me occurred in an infant ten weeks old. Of 111 cases reported by Councilman, 29 occurred in infants and children. Of a series of 70 cases of cerebrospinal meningitis reported by me, 47 per cent. were under two years of age; the youngest was four months of age, and 61 per cent. of the cases were under four years of age. The oldest child in my hospital service was fourteen years of age. Thus the average age was two years.

Morbid Anatomy.—In certain sporadic cases of cerebrospinal meningitis of the epidemic type the clinical symptoms may have been very marked, and yet post-mortem examination fails to reveal any gross mircoscopical lesions of the brain and pia mater. They appear to be normal. Under the microscope, however, a slight infiltration of the pia with pus and fibrin and a new growth of cells is seen. In other cases there is an extensive infiltration of the pia with serum, fibrin, and pus. The exudation is especially profuse at the base of the brain and on the posterior surface of the cord, more especially in those cases which will hereafter be described as posterobasic meningitis. The ventricles of the brain may be markedly distended with serum and even pus. Among the associated lesions found are subserous punctate hemorrhages of the endocardium; ecchymoses and petechiæ of the skin, hyaline and granular degeneration of muscle, multiple abscesses of the skin, suppuration of the joints, parenchymatous degeneration of the heart, liver and kidneys, and swelling of the lymph-nodes and spleen. In all the epidemic cases of the type referred to in this section the Diplococcus intracellularis is found in the exudate of the pia mater and cortex of the brain and in the fluid of the ventricles.

Symptoms.—There are certain types of cerebrospinal meningitis which are seen both in the epidemic and sporadic forms of the disease. The malignant types are seen rather in the epidemic forms; whereas the milder types are seen in the sporadic cases. Clinically, therefore, we may divide all cases of epidemic cerebrospinal meningitis into three forms: The first form is the malignant type of the disease, in which the children, in previous good health, are attacked and die within twenty-four or thirty-six hours of the onset of the disease.

The following case, one of the first of the epidemic of 1904, is a characteristic example of this type: An infant twelve months old, nursed at the breast; perfectly formed, large, healthy, bright child, never previously affected by any illness, nursing, and bowels normal. On the morning of the onset of the illness the child appeared drowsy

PLATE IX.



Cerebro-spinal Meningitis (Meningococcus). Rigidity of the neck, opisthotonus, characteristic position of the arms. Infant eight months of age; fatal issue.

polyuria. The spleen may be enlarged. The type of cases just described corresponds to the mass of cases seen in an epidemic.

A third type of this disease is more puzzling in its character; it affects infants and young children in apparent health. Infants and children are noticed to have a constant rise of temperature; there may be vomiting; there is restlessness; if nursing, they refuse the breast. The fever after a few days takes an intermittent course, mounting as high as 104° and 105° F. at certain times of the day; falling to the normal or subnormal at others. In the intervals of freedom from temperature the children or infants will play, and when the temperature rises they complain of headache (if old enough), become drowsy and irritable, refuse nourishment, and develop symptoms which point toward meningeal inflammation, such as the Kernig symptom, rigidity of the back of the neck. In these cases the typical symptoms of meningitis are not always present. Delirium may not be constant or may not extend over the twenty-four hours. The rigidity of the neck may not be very marked, especially in young infants, as has been intimated. The Kernig symptom in children, especially below two years of age, may not be evident. The most characteristic feature of these cases, it seems, is the prolonged temperature of an intermittent type, closely resembling malarial fever. In fact, many of these cases have been mistaken for malaria.

There is a fourth type of case, which will be described under the heading of Postero-basic Meningitis, which is observed not only sporadically, as has been remarked by Still, but also in epidemics.

Individual Symptoms.—MODE OF ONSET.—In all the cases that I have had an opportunity to observe in my hospital and private practice, and in which the diagnosis was confirmed by lumbar puncture, the main characteristic of the disease was its sudden onset. In only a small percentage of cases was there a doubtful history of sudden onset. In this respect the disease differs markedly from other forms of meningitis, especially those of the tuberculous type, in which the invasion is slow and insidious. From a study of the symptoms the onset may simulate an attack of gastro enteritis in some children.

Cerebral Symptoms.—If the fontanelle is not closed there is distinct bulging or tenseness, even in the early stages of the disease, certainly before the fifth day. The patients suffer from delirium or coma, and in the milder cases headache is the principal symptom, and periods of consciousness alternate with those of stupor. Rigidity of the neck, either slight or marked, is present at one time or another in all cases, and opisthotonos is present in about 70 per cent. of the cases.

According to Osler, neck rigidity or opisthotonos was not present in the adult form of primary pneumococcus meningitis. In one case, however, of my own, of primary pneumococcus meningitis in a child, neck rigidity was present. There is hyperæsthesia of the surface, and the patients cry out if the bed is jarred or the skin touched. In some cases there are recurrent rigors and convulsions, either unilateral or general. There may be facial paralysis and hemiplegia in the early or the later stages of the disease.

Reflexes.—In the majority of cases of epidemic cerebrospinal meningitis the patellar reflex is present in the early stages of the disease, but it may disappear in the rapidly fatal or moribund cases. The so-called tâche cérébrale of Trousseau is obtained in all cases.

BABINSKI REFLEX.—Babinski, a French neurologist, described the extension of the great toe and separation of the other toes on irritation of the plantar surface of the foot as a characteristic sign of disease of the pyramidal tracts or the lateral columns of the cord. In epidemics of cerebrospinal meningitis this phenomena is obtained in only a small percentage of cases, in contradistinction to what is noted in the tuberculous form of meningitis, in which it is common, being obtained in 6 of 26 of my cases of tuberculous meningitis. The Babinski reflex is of very little value in children and infants below two years of age, for a phenomenon closely resembling it is obtained in perfectly normal individuals at this age.

Kernig Symptom.—The Kernig symptom—that is, an inability to extend the leg on the thigh when the latter is flexed on the trunk—is obtained at one time or another in all cases of cerebrospinal meningitis. In children below two years of age, however, this sign must be accepted with caution because of the natural tendency in infants and children of this age to contraction of the lower extremities, a variety of normal myotonia (Fig. 46). On the other hand, in cases of so-called cerebral symptoms complicating pneumonia and typhoid fever, the Kernig phenomenon may also be apparent, so that, although it is present in all cases of meningitis, it is not pathognomonic of the disease. It may be absent in cases of the malignant type in which there are collapse symptoms.

Hyperæsthesia.—In the majority of cases cerebrospinal meningitis, after the symptoms are fully established, the patients are irritable, refuse to be comforted, start at the slightest sound, lie mostly on the side, the arms and lower extremities flexed, the body taking a crouching position. Any attempt to disturb the patients is met with resistance. The amount of hyperæsthesia varies not only in the different epidemics, but in different types of the disease, but it is present in most cases, thus being in marked contrast to what is seen in the tuberculous form of meningitis, in which the children lie in a stuporous condition, do not notice their surroundings, cannot be roused, and are not as irritable as in the epidemic cerebrospinal form.

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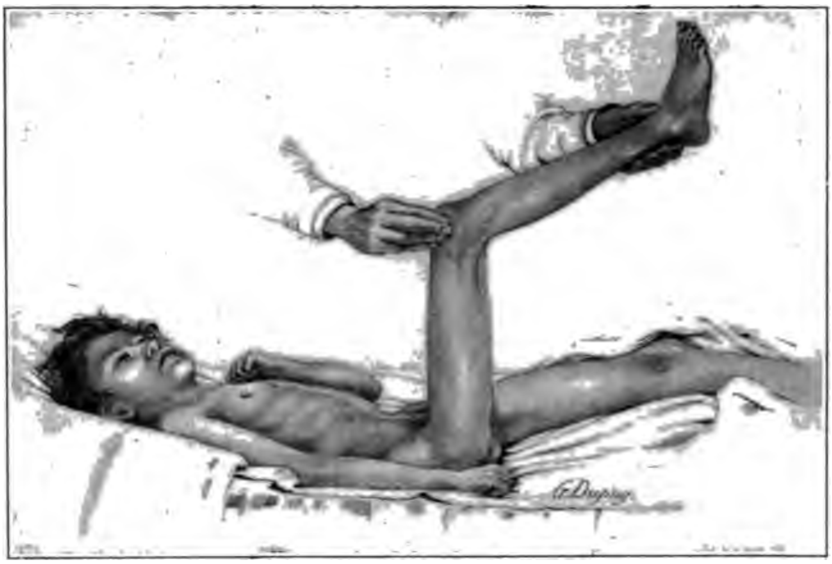
MacEwen's Sign.—MacEwen has shown that in children, in various forms of meningitis, percussion of the skull over the anterior horn of the ventricles will give a tympanitic note if the head is so held that the frontal or parietal bone may be percussed over the anterior horn of the ventricle. The patient is placed in the sitting posture, with the head inclined to one side, and percussion of the inferior frontal or parietal bone is carried out.

The MacEwen sign is obtained in those cases of the cerebrospinal meningitis in which there is an accumulation of fluid in the ventricles, and was present in only 2 cases of 13 studied with a view to obtaining this sign. It is more common in the tuberculous forms of meningitis.

FIG. 46.



Kernig symptom in a case of cerebrospinal meningitis of the epidemic type. Female, nine years of age.

Facial Paresis.—In epidemic cerebrospinal meningitis facial paralysis may occur in the very severe cases at the outset of the disease, especially if the base of the brain is involved.

Paralysis.—There may be paralysis not only of the facial muscles, but of the extremities on one or the other side, either at the outset of the disease or toward the close.

Eyes.—There may be an initial conjunctivitis, keratitis, strabismus, contraction, dilatation, or inequality of the pupils; neuritis of varying grades of the disk; atrophy, and finally purulent choroiditis. There is no appreciable impairment of vision in some cases. In a four-months-old baby paralysis of the orbital muscles of one side appeared early in the disease. A peculiar phenomenon has been observed by me and described by others referable to the pupils: If an attempt is made to bend the head forward as the patient lies in bed unconscious, the pupils will be observed to dilate (mydriasis).

Contrary to the generally accepted opinion, we have found that expert examination of the fundus of the eye in cases of cerebrospinal meningitis of the epidemic type revealed few changes in the optic pupilla in the majority of cases. In some cases there was dilatation of the veins, or congestion without neuritis. In only one case was there descending neuritis. This corresponds very closely to what Barlow and Gee found to be true both of the vertical and postero-basic forms of meningitis. In a group of 26 cases of meningitis of the tuberculous variety, however, examined by an expert ophthalmologist, some change was found in the fundus in fully 77 per cent. of the cases. This change consisted either of an optic neuritis or papillitis, or the presence of tubercles in the choroid.

Blood.—The leucocyte count in cases of cerebrospinal meningitis of the epidemic type ranges from 20,000 to 55,000 to the cubic millimetre in 55 per cent. of the cases. There are cases, however, with a low leucocyte count of 11,000 to 12,000 to the cubic millimetre. This corresponds very closely to what was found by Osler to be true of the adult cases. In tuberculous forms of meningitis, however, of infants and children, in 40 per cent. of the cases there is a leucocyte count of 20,000 to 25,000 to the cubic millimetre, and in 60 per cent. of the cases the leucocyte count is below 20,000 to the cubic millimetre. Rarely, however, does the leucocyte count exceed 24,000.

In the fatal cases, in which the lumbar puncture may yield a fluid markedly purulent, the leucocyte count may mount from 35,000 to 55,000 to the cubic millimetre. On the other hand, a fatal case with fluid obtained by lumbar puncture might show a leucocyte count not exceeding 23,200.

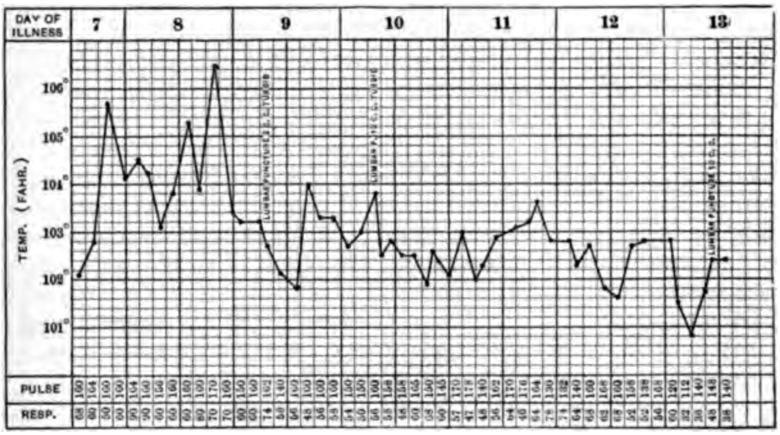
Cases which have recovered may show in the course of the disease a leucocyte count of 14,000 to 28,000 to the cubic millimetre, and they may have mounted as high as 45,000. It cannot, therefore, be said that a prognosis as to recovery or fatal issue can be made from the leucocyte count alone in cerebrospinal meningitis.

Pulse.—The pulse in cerebrospinal meningitis, as a rule, is rapid and irregular; but there are periods in which the pulse is slow, sometimes 80 or even lower. This is not as common, however, as the rapid pulse.

Respirations.—The respirations, as a rule, are shallow, increased in frequency, and irregular in rhythm. In a few cases there may be Cheyne-Stokes respiration. In other cases Cheyne-Stokes respiration is not seen in the whole course of the disease; as the fatal issue approaches, the respirations may cease before the heart ceases to beat. In the terminal stages the respirations sometimes fall to 10 a minute, and the pulse to 50, indicating the onset of general paralysis.

Temperature.—There is no curve of temperature which is distinctive of cerebrospinal meningitis. It may be said, however, that the temperature in many cases is of the intermittent variety, and for this reason these cases are frequently mistaken for malaria. In the intermittent type of temperature the remissions are very great, sometimes ranging eight degrees in twenty-four hours; that is, a temperature which has been high will in a few hours fall to the subnormal to rise again. This is not uncommon and may extend over weeks. On the other hand, the temperature may remain persistently high, especially in the rapidly fatal cases of the malignant type.

FIG. 47.



Cerebrospinal meningitis. Female infant, eight months of age; unconscious on admission; fatal issue. (Meningococcus.)

In the chronic cases the temperature may fall to and continue within normal limits for days or even weeks. In some cases, after the temperature has remained normal for days or weeks, there may be a so-called recrudescence of temperature of an intermittent type extending over a week or more. This does not preclude ultimate recovery. In one case in the recent epidemic the temperature continued of the intermittent type, with the remissions mentioned above, for eight weeks, fell to the normal for a week, rose again, continued intermittent for a week, and finally fell to the normal and remained there. In this respect the temperature may even resemble typhoidal curves of the third or fourth week.

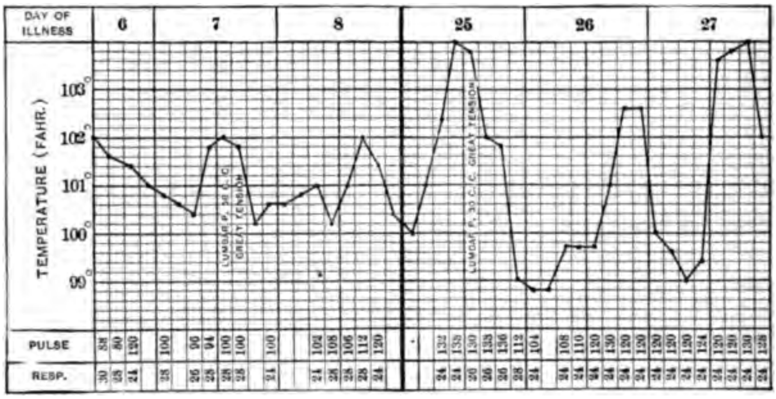
Spleen.—The spleen may be enlarged in some cases.

Ear.—The ear may be the seat of otitis or mastoiditis. Deafness, especially where the base is involved, may supervene very early.

The **anterior fontanelle,** as has been mentioned, in infants and children in whom the structure has not closed, may be tense or distinctly bulging; and in those cases in which there is considerable accumulation of fluid the posterior fontanelle may reopen.

Skin.—In many of my cases there has not been that prevalence of skin rashes described by most authors. It has been only in the last epidemic of 1904 in which skin eruptions were prevalent. They included the roseola resembling that of typhoid fever. The roseola appears, as a rule, at the outset of the disease, and may recur in the course of the disease. Purpuric spots extending over the general surface are common at the outset, as well as ecchymoses, and these may disappear within a few days, leaving absolutely no trace of their presence; or recurrent crops of ecchymoses and petechiæ may appear in the course of the disease. Herpes labialis varies in different epidemics as to its frequency, being absent in the majority of cases in some epidemics, and being frequent in others. Herpetic eruptions may occur elsewhere on the trunk or extremities. I have seen extensive herpes on the hand. One case has come to my notice in which the herpes were quite generally distributed over the trunk and extremities.

FIG. 48.



Cerebrospinal meningitis. Female child, eight years of age; temperature at two extremes of the illness. Recovery. (Meningococcus.)

Complications.—In some epidemics of cerebrospinal meningitis there are few complications. Those cases which recover do so with very little to show that the nervous system in any of its extent has been severely compromised. The eyesight is not injured, nor is there subsequent hydrocephalus in any cases. In other words, the recoveries when they occur are complete and satisfactory. This is especially true of small epidemic outbreaks occurring over the course of years. In the recent epidemic of 1904, however, the complications were more frequent; joint complications were observed in 2 cases of a series of 30; blindness was not an uncommon complication, as also deafness. Recovery was incomplete, with hydrocephalus in several cases of a series of 30. Pneumonia was observed as a complication of cerebrospinal meningitis of the epidemic type in 2 fatal cases.

Sequelæ.—Recovery may take place without compromise of any of the senses or functions of the patient. Both in young and older children hydrocephalus, either of a mild or severe type, may supervene in the course of the disease; it may run a short course and the patient recover with a mild form of hydrocephalus, which in years gives rise to nervous symptoms, such as partial paresis or epileptic form of convulsions. Severe types of hydrocephalus lead in many cases to permanent idiocy or imbecility, with or without paralysis. In some cases blindness or deafness results as a direct cause of meningitis. Arthritis, which sometimes complicates the disease, has a tendency to get well and leave no marks of its presence. Many patients recover with so-called sensitive spines, or paresis of certain sets of muscles, which later in life becomes apparent.

Optic neuritis or blindness occurring in the course of the disease very frequently retrogrades, and the patients, on recovery, bear no marks of any ocular lesion.

Characteristics of the Fluid Obtained by Lumbar Puncture.—The fluid obtained by lumbar puncture in cases of meningitis, studied both as to cytology and bacteriology, is of particular interest as regards the possibility of making a diagnosis. The cytology of the fluid obtained in cerebrospinal meningitis shows a preponderance of the polynuclear leucocytes. In a small percentage of cases the mononuclear cells, contrary to the general belief, may be prevailing elements, thus closely resembling what is seen in tuberculous meningitis. In chronic cases mononuclear leucocytes abound; and in these cases, especially those of the basic type described by Still, the cytological picture resembles that of tuberculous meningitis. The fluid obtained by lumbar puncture in cerebrospinal meningitis may be quite clear, with scarcely any sediment, and may be markedly purulent, in this respect differing from the fluid obtained in tuberculous forms of meningitis, which is clear in at least 70 per cent. of the cases.

Bacteria.—In the vast majority of cases of cerebrospinal meningitis the Diplococcus meningitidis intracellularis of Weischelbaum was found at one time or another, either in leucocytes or outside of the leucocytes. In the chronic cases, however, there are times in which the Diplococcus intracellularis is not found. This is especially true of the posterior-basic cases. In those cases in which the diplococcus has not been found during life in the fluid obtained by lumbar puncture, it may be discovered post mortem in the fluid obtained from the ventricles of the brain.

Course of the Disease.—The course of the disease after the symptoms are fully developed in typical cases has been indicated in the first part of this article. The patient lies unconscious, the head is retracted, and in some cases the back arched. The delirium is constant, and the patients complain of headache. The neck is rigid; some patients complain also of pain in the course of the sciatic nerves. When disturbed they cry out with pain. There may be rigors, during which the patients become cyanosed and the heart feeble. The respirations are shallow and irregular. If the case lasts over a week the patients may refuse nourishment, and on this account marked emaciation sets in. In some cases the disease takes on an abortive type. After a period of headache, fever, vomiting, intervals of remission of all symptoms, including temperature, alternate with intervals in which the temperature runs an intermittent course, with a return of the headache, stupor, and uneasiness, convalescence finally sets in, and the patients rapidly recover. Other cases result fatally in a few days. Some cases run a course of from eight to fifteen weeks, with the temperatures described, great emaciation, and finally make an incomplete recovery. Others attain a freedom from symptoms, but emaciation and paralysis persist, or even blindness and deafness, until an intercurrent affection ends the sufferings of the patient. As will be shown, there are few recoveries in children below two years of age. In other cases recovery takes place, but idiocy, hydrocephalus, blindness, or palsy may persist.

Diagnosis.—Cerebrospinal meningitis must be differentiated from tuberculous meningitis, typhoid fever, and pneumonia with cerebral symptoms.

It is distinguished from tuberculous meningitis by the sudden onset, its continued or intermittent higher febrile movement, the early onset and marked rigidity of the neck and opisthotonos, and, as has been intimated, the higher leucocytosis, and finally by the examination of the fluid obtained by lumbar puncture. Cerebrospinal meningitis is distinguished from typhoid fever by the fact that in the latter disease there is a leucopenia and a constant enlargement of the spleen with Widal reaction. On the other hand, there may be cases of typhoid fever in which the cerebral symptoms are very marked and in which a meningitis may be present, due to an invasion of the meninges of the brain and cord by the typhoid bacillus. In this set of cases the diagnosis will be very difficult without the aid of a lumbar puncture. The latter procedure being entirely innocent in its nature, should be made in order to exclude the severer affection of cerebrospinal meningitis. A pneumonia with cerebral symptoms will at the outset closely resemble a cerebrospinal meningitis, especially in very young children. Even if an examination of the lungs reveals a pneumonia during an epidemic of meningitis, we cannot always exclude the latter disease without resort to a lumbar puncture, for cases of meningitis of the epidemic cerebrospinal type caused by the intracellular diplococcus are met in which pneumonia is present as a complication. On the other hand, pneumonia *per se* with cerebral symptoms does not, as a rule, give us the very marked rigidity, opisthotonos, petechiæ, intense cephalalgia, and Kernig symptom seen in cerebrospinal meningitis. I have, however, met isolated cases, both of pneumonia and typhoid fever with cerebral symptoms, in which a Kernig symptom was obtained, as well as the so-called tâche cérébrale of Trousseau, although these cases are certainly exceptional; in any doubtful case we should not hesitate, as has been said, to resort to lumbar puncture in order to clear up a given case.

Prognosis.—The mortality of cerebrospinal meningitis varies largely with the severity of the infection and in different epidemics. In some epidemics the malignant cases seem to predominate; that is, those cases which die within a short time (from twenty-four hours to five days) after the onset of the disease. On the other hand, in small epidemics the mortality may not exceed 48 per cent. There are epidemics in which the mortality has risen as high as 90 per cent. Especially fatal are the posterior-basic cases and those attended by malignant features at the very outset of the disease. The prognosis, unfortunately, cannot be predicted in cerebrospinal meningitis, either from the nature of the fluid obtained by lumbar puncture, or from the condition of the blood as reflected in the leucocyte count, or the range of the temperature. We can only say that it is especially fatal the younger the patients. We have records, however, of cases of cerebrospinal meningitis occurring in infants of five months and one year of age, substantiated by culture and lumbar puncture, in which recovery occurred.

The **treatment** of cerebrospinal meningitis is symptomatic. There have been many suggestions and variations of treatment in different epidemics; but it must be said that the more we see of this disease, the more we are impressed with the fact that it is self-limited, runs its course within a certain time, and can only be treated by palliative measures.

The nutrition of the patient is a most important element in the management of these cases. In those cases in which the patient is comatose and refuses to take nourishment by the mouth, it is a difficult problem to maintain the nutrition of the patient. In many cases nourishment must be given by the rectum, and in some must be introduced into the stomach by means of gavage. In the first case we frequently find that after nourishing the patient by the rectum for a few days this viscus becomes intolerant and very little nourishment is retained. Peptonized milk and somatose in the form of enemata are the most available forms of nourishment by the rectum. Gavage does not meet our ideals as to nourishment of the patient, because there is resistance to this procedure on the part of the unfortunate sufferers. Thus, each individual case will be a problem to the physician; some patients take food with avidity, and in these cases milk and broths are the principal forms of nourishment given.

The bowels of these patients are generally constipated, and from time to time a cathartic must be given; the most preferable cathartics are the mercurials, calomel in dose of ½ to 2 grains, are given to clear the bowels. This may be repeated at intervals of forty-eight to seventy-two hours. Enemata do not seem to reach the majority of cases. The headache is very severe in a great number of cases, and no remedy that we know of completely relieves the symptom. Morphine given in moderate doses relieves some patients. In others this drug is not well borne, and the patients seem to become more stupid and the circulation weaker under its continued use. The author has tried the various drugs of the coal-tar series.

Pyramidon in doses of 5 to 7 grains, given at intervals of three to four hours, seems to have relieved a certain percentage of cases. The head is shaved and the ice-cap applied. Even this procedure is not well borne by some patients, and they strongly protest against it. It seems to increase the pain.

Following the English school, the iodide of sodium or potassium is given in liberal doses to limit the formation of exudate. It is impossible to say whether we can be successful by this means in influencing the inflammatory processes. However this may be, the author has given these patients liberal doses of iodide of sodium or potassium, preferably the former. Children under one year of age will bear 1 to 2 grains of the iodide of sodium three or four times daily without serious disturbance of the functions of the stomach.

The delirium is treated with liberal doses of mixed bromides of sodium, potassium, and ammonia. In some cases chloral in moderate dose is added to this mixture, and is well borne by the patient. It does not depress the circulation.

The irregularity of the heart which is present in a large number of cases does not call for any active treatment. Alcoholic stimulants should be avoided if possible, as there seems to be no indication for their use. ·One of the principal modes of meeting restlessness, the occasional high temperature, the rigors and accompanying cardiac weakness, is the systematic use of warm baths. The patients are placed in a warm bath of a temperature of 105° to 107° F. three times in the twenty-four hours. Care should be taken to lift the patient gently from the bed into the bath. Massage should not be performed as in the ordinary bath given in pneumonia, while the patient is in the bath, inasmuch as this friction irritates and excites the patient and seems to cause a great deal of pain. The duration of the bath should be from five to ten minutes. The time for giving it should be chosen when the temperature is on the rise, the irritability of the patient at this time being greatest. If the heart should become very weak, camphor is indicated; if possible, by the stomach. If this is not feasible, camphor, in the form of camphorated oil, should be given subcutaneously.

In cases in which there are symptoms of increasing exudate in the subarachnoid space, such as extreme rigidity, opisthotonos, coma, delirium, bulging fontanelle; in young infants, chills with subsequent rises of temperature, the operation of lumbar puncture should be performed. In those cases in which coma and delirium supervene at the very outset of the disease, lumbar puncture may be performed within twenty-four to forty-eight hours of the onset of symptoms. I have frequently withdrawn in such cases 40 to 50 c.c. of a turbid fluid, with subsequent relief of delirium and headache. We should not hesitate after the first puncture to repeat the procedure within forty-eight hours if symptoms either recur or remain stationary. In young infants and children especially repeated lumbar puncture seems to be called for by the very fact that in these subjects the continued pressure and increase of fluid in the subarachnoid space and in the ventricles of the brain increases the tendency to dilatation of the ventricles, a serious complication which may lead to collapse symptoms, sudden death, or ultimate chronic hydrocephalus. In those cases in which at the outset of the disease the head retraction is very marked, the lumbar puncture is sometimes unsatisfactory, inasmuch as little fluid is withdrawn. In these cases the exudate at the base of the brain and the extreme retraction of the head may cut off the communication of the subarachnoid space and spinal cord with the ventricles of the brain. The canal of Majendie, through which this communication is sustained, is in these cases occluded.

Lumbar puncture in a certain proportion of cases presents no relief to the patient; nor can it be said, as yet, that lumbar puncture is curative. As has been intimated, it relieves symptoms of headache and delirium. It removes a certain amount of purulent exudate which is a menace to the vital structures of the brain and cord, and is thus a method of drainage rather than a curative measure. It may, in cases of sudden distention of the ventricles of the brain with fluid, avert death.

The amount of fluid withdrawn at each puncture should be from

20 to 50 c.c., depending greatly on the extent of pressure present, as indicated by the manner in which the fluid flows from the puncture cannula. If the fluid flows drop by drop, a small amount, 20 to 30 c.c., is withdrawn. In some cases the fluid fairly spurts from the cannula, and in such cases 50 c.c. may be withdrawn, or even more. In other cases the exudate is so thick and purulent that it will not flow from the cannula except in large, thick drops at long intervals. We should not in these cases attach a syringe to the cannula and apply suction to the fluid, for in this way, it has been shown, hemorrhages may be caused in the spinal cord and in the pia of the brain. It need scarcely be mentioned here that everything in connection with the operation of lumber puncture should be carried out with due regard to asepsis. The puncture wound may be closed with collodium, or with a dry, sterile dressing of a few layers of gauze, held in place with plaster, and an anæsthetic is not needed in children but may be administered to the adult. As might be supposed, a number of modifications on the procedure of simple lumbar puncture have been proposed. In the epidemics occurring in Portugal and Spain, lysol in 1 per cent. solution has been injected through the cannula into the subarachnoid space after a certain amount of cerebrospinal fluid had been withdrawn. One per cent., 2 per cent., and in New York as much as 10 per cent. solutions have been injected, the solutions being at first carefully sterilized. It cannot be said that any success has followed injection of drugs, either lysol or preparations of mercury, in cerebrospinal meningitis. Their use has not been followed by a reduction of mortality. Thus, there is no specific treatment of cerebrospinal meningitis which will abort the disease, shorten its duration, or cure the patient.

Acute Lepto-meningitis.

(*Vertical Meningitis.*)

In this form of meningitis the vertex or superior surface of the brain is affected; the region of the cerebrospinal foramen may escape, but not necessarily so, and in some cases the base also may be affected.

Occurrence.—It is found in the newborn and children as a complication of sepsis, erysipelas, pneumonia, influenza, diseases of the ethmoid and mastoid bones, perforation of the bones of the skull, or suppurations elsewhere, such as retropharyngeal abscess.

Etiology.—The essential cause is an invasion of the tissues of the meninges of the brain by streptococci, pneumococci, the intracellular diplococcus of Weichselbaum, the influenza and coli bacilli.

These cases are sometimes difficult of diagnosis, because in many of them the classical symptoms of meningitis are absent. In the early stages of the disease anatomically there is dryness and opacity of the pia hyperæmia. Later, œdematous conditions of the pia supervene with the formation of lymph and fibrin along the sulci and in the tissue of the pia mater and on its surface. Later, the purulent exudate may extend over the surface of the brain, involving not only the base of the brain, but also the spinal cord. In some cases the exudate does not penetrate the ventricles of the brain; in others inflammation extends into the ventricles. In this form of meningitis there are complications either primary or secondary, such as pneumonia, empyema, pericarditis.

Symptoms.—The diagnosis is difficult. The symptoms are often latent. Retraction of the head is very often absent, and ocular symptoms are rarer; in fact, the fundus in many cases is found to be normal. Vomiting is less frequent than in the basic forms of meningitis to be described, or in the cerebrospinal forms just described. Convulsions of a violent character may be present; they may be repeated throughout the disease, and are associated in some cases with high temperature; in other cases they are absent. These convulsions may be epileptiform. Clonic spasms may be local at first, but, as a rule, they become bilateral and general. There may be, as in meningitis, tonic spasms. The duration of the disease is shorter than in posterior-basic meningitis, may last from one to two days to as many weeks, and in exceptional cases may become chronic. In many cases it is impossible, unless a lumbar puncture is made, to differentiate these cases from tubercular meningitis. Nor is it possible, if the exudate extends to the spinal cord and rigidity sets in, to differentiate a so-called vertical case from an ordinary cerebrospinal meningitis of the epidemic type unless a lumbar puncture is made. The differentiation, therefore, of these cases must depend on a continued observation of the case and the performance of lumbar puncture.

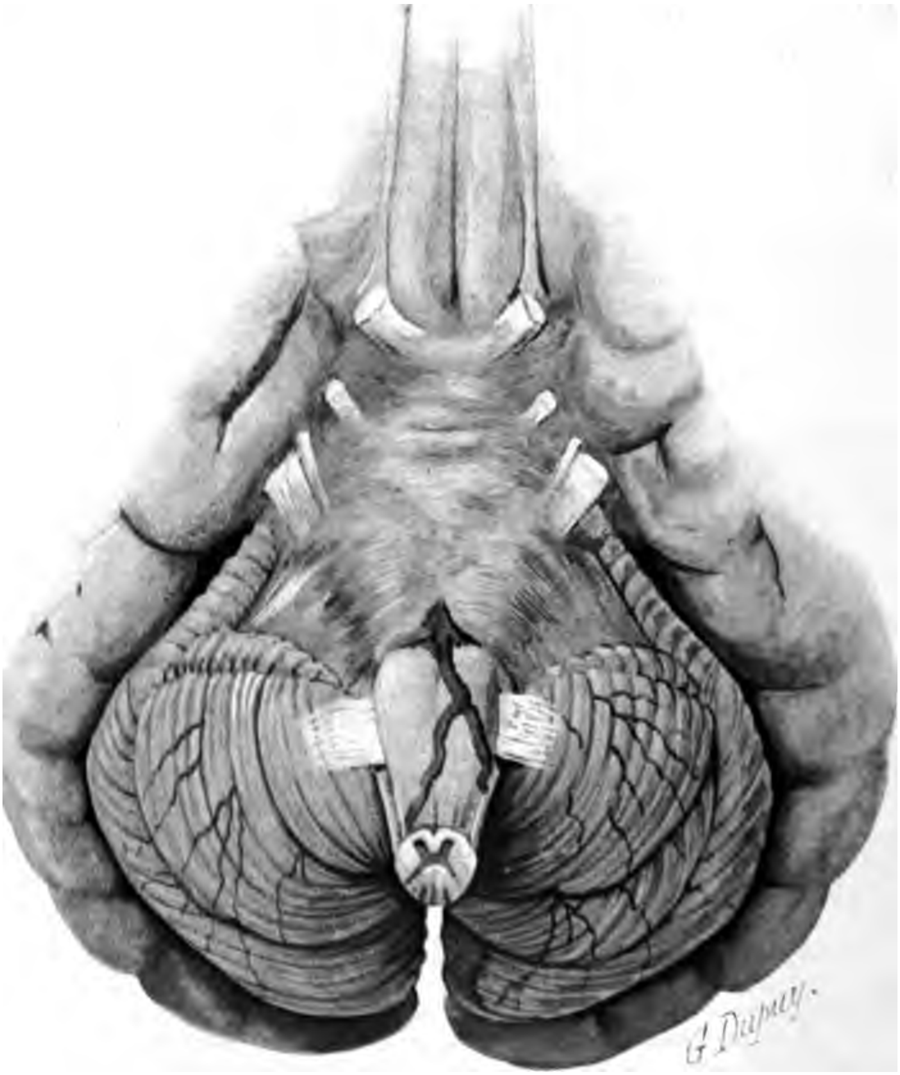
Posterior-basic Meningitis.

Posterior-basic meningitis is so called because the inflammation affects the posterior part of the base of the brain and the structures in this location, and rarely spreads to the vertex of the brain, at most only affecting the tips of the temporosphenoidal lobes, and in some cases extending forward to the optic commissure. These cases were first described by Gee and Barlow, in the *Bartholomew Hospital Reports* of 1878, and subsequently by Still in 1898.

Occurrence.—The affection occurs in infants and children below the age of two years, and is rarely seen in older children. I have seen exceptional cases in children of three and five years of age.

Etiology.—These cases, according to Still, are caused by a

PLATE X.



Posterior Basic Meningitis. (Gee, Barlow and Still.)
Author's case.

diplococcus which is identical with the diplococcus of Weichselbaum, Jäger, and Leichtenstern, an intracellular diplococcus not staining with Gram's method. Although Still thought that these were only sporadic cases of the epidemic form of cerebrospinal meningitis, it can now be said that they are seen very frequently and in large numbers in epidemics of cerebrospinal meningitis, and may occur sporadically. They are only specific, inasmuch as they are a form of cerebrospinal meningitis as it occurs in younger children and infants. These cases divide themselves into those which are fatal after six weeks; those which die after three or four months with hydrocephalus; and those which recover. In the first set of cases anatomically we find pus and lymph at the base of the brain and extending down the cord. In the second set of cases there is simply thickening of the pia and arachnoid, with adhesions between the cerebellum and medulla. The inflammation may spread down the cord to a varying degree, and upward along the lining membrane of the ventricles, and afterward along the base as far as the optic commissure. In the chronic cases there may be adhesions of the meninges either in the anterior part of the base of the brain or even on the vertex, showing that this has been slightly involved. The adhesions at the base may unite the medulla and cerebellum and obliterate the foramen of Magendie or the fourth ventricle. This results in accumulation of fluid in the ventricles with hydrocephalus. In some cases the ventricular fluid is clear; in others it contains flakes of fibrin and pus.

As has been shown, complications in this form are rare. Occasional arthritis is found. In some cases Still has found tuberculous foci; of the viscera, which he considers accidental. In other cases the middle ear may contain mucopurulent secretion, but no evidence of the extension of the ear disease to the brain or meninges.

Symptoms.—The onset, as in cerebrospinal meningitis, is abrupt and has the same symptomatology. The most characteristic symptom clinically of these cases is the retraction of the head. This supervenes early and continues until death or recovery of the patient. Convulsions, tonic or clonic, occur early in the disease, but are less frequent than in meningitis, involving the superior surface of the brain and cord. There are rigidity of the limbs and opisthotonos, and an increase and diminution of this rigidity, in the course of the disease, with tetanic contractures of the upper and lower extremities, as shown in the accompanying drawings. Vomiting is one of the first or early symptoms, and may occur throughout the disease. After the disease has lasted some time the eyes have a fixed stare; there may be strabismus or nystagmus; the pupils are contracted, or later may be dilated. Optic neuritis is not common, though the patients may be blind. If the anterior fontanelle is still open, it bulges with the increasing hydrocephalus, and in some cases the posterior fontanelle, which may have been closed, is reopened, the sutures become widely separated, and the children finally lay unconscious and perform automatic movements with the facial muscles, mouth, and extremities. The rigidity and retraction in some cases are extreme; the opisthotonos is very marked at times; at others the neck rigidity will relax, but on the least irritation, either of friction or otherwise, the opisthotonos and tetanic spasms recur. Recovery may take place with retrograde of most or all of these symptoms, or imperfectly so with hydrocephalus. In some of these cases the temperature curve at first is high, and after the disease has lasted some time it may drop to the normal and remain there, or rise a degree above the normal, at times thus simulating tuberculous meningitis; or the temperature may be normal for periods of weeks and then suddenly, without any apparent cause, show wide variations, with high temperatures during certain parts of the day and subnormal temperatures at others. Death may supervene suddenly without cause.

The **prognosis** is very bad. There are few recoveries, and in an epidemic most of these children die or develop an incurable hydrocephalus.

The **treatment** is directed toward the relief of the hydrocephalus. As soon as this is established or evident, repeated lumbar puncture should be performed, in order to stay the increase of the fluid in the ventricles, and, if possible, effect a cure of the hydrocephalus; this is not always possible. The treatment of the symptoms are the same as that laid down for cerebrospinal meningitis.

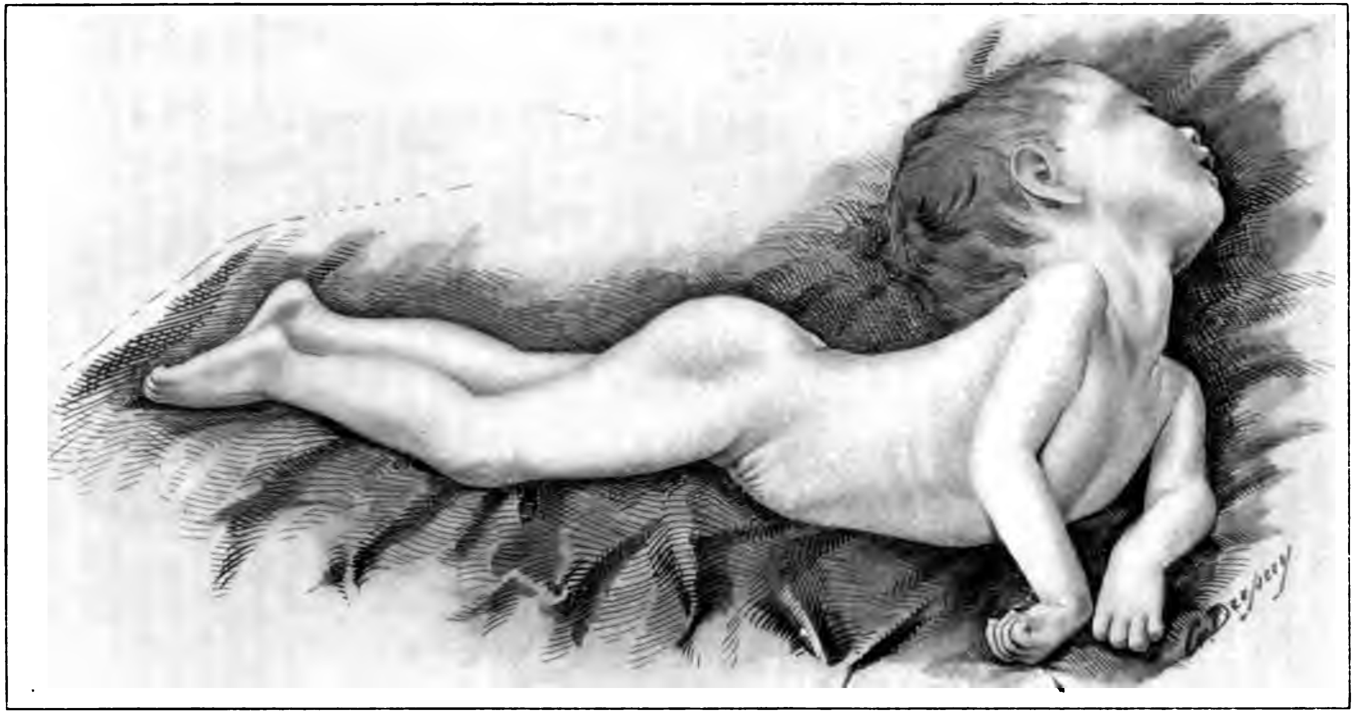
Meningitis Serosa (QUINCKE).

(Acute Internal Hydrocephalus.)

Meningitis serosa, or acute internal hydrocephalus, must not be confounded with tuberculous meningitis, which formerly was called acute internal hydrocephalus. Meningitis serosa was described in 1893 by Quincke. Four years later Bönninghaus reported some of these cases, and since then a number have been described in the literature.

Occurrence and Definition.—It is a comparatively rare disease, and occurs only between the ages of one and five years. It consists of a serous inflammation of the extra- and intra-cerebral pia mater, and as a consequence of this inflammation there is an inflammatory œdema in the subarachnoidal space, accompanied by acute internal hydrocephalus, or serous exudate in the ventricles of the brain. We have two forms of this condition: in one the brain and membranes are found to be the seat of inflammatory œdema, in which the exudate in the ventricles is comparatively small in amount; in the other, the more common form, there is a very large exudate in the ven-

PLATE XI.



Posterior Basic Meningitis. Characteristic position of the head and extremities.
Child aged fourteen months.

tricles, and the membranes of the brain and pia mater are but little affected.

The **etiology** is not quite clear. Quincke insists that the condition may occur idiopathically, in a manner similar to an idiopathic pleurisy. Later authors are inclined to regard serous meningitis, however, as an infectious process, due to the invasions of staphylococci or streptococci, which are found in the ventricular fluid removed through lumbar puncture or post mortem. Some of these cases may follow a chronic hydrocephalus; others may be traumatic or complicate an acute febrile disease, such as typhoid fever or pneumonia.

The **symptoms** are not always marked, and it is not always possible to recognize the disease with certainty. The differential diagnosis from other forms of meningitis, such as the tuberculous form, is made with the greatest difficulty. The disease may begin with varying symptoms. The children are peevish and restless; they refuse to take nourishment. There may be constipation, disturbances of the process of digestion, and finally vomiting, with continued emaciation. The temperature in all cases thus far observed is raised but little above the normal; or, if raised to 103° F., rapidly falls again to normal. The pulse may be normal or slightly increased in rapidity. A constant symptom in children below fourteen months is that the head increases in circumference, the sutures are forced apart, and the anterior fontanelle becomes tense and bulging. The cerebral symptoms consist mostly of sopor, uneasiness, strabismus, and nystagmus. Sooner or later convulsions appear, involving most of the musculature or groups of muscles. In some cases an early optic neuritis has been observed.

The course of the disease is a protracted one, inasmuch as the symptoms may extend over weeks or months, ending finally in death, preceded by an increasing cachexia.

In those cases which have recovered, the circumference of the head has returned to its normal dimensions.

Morbid Anatomy.—The most striking lesions found post mortem are a dilatation of the ventricles of the brain with an increased amount of intraventricular fluid, by which the surface of the brain is compressed and the convolutions flattened. The ependyma is swollen, thickened, and the surface granular. The choroid plexus is hyperæmic. The membranes of the brain may be dull and more or less hyperæmic. In some rare cases, at the base of the brain a circumscribed purulent meningitis has been described, which supports the view that serous meningitis may follow a localized condition of this character.

A characteristic of serous meningitis is the cloudy swelling with proliferation and desquamation of the cells of the ependyma, and cellular infiltration of the brain substance beneath the ependyma with round cells, especially along the bloodvessels. In such cases there is really an ependymitis or meningitis ventricularis.

The **diagnosis** of serous meningitis must be made from meningitis of other varieties, especially of the tuberculous or cerebrospinal type. The author is inclined to believe that during life a very careful exclusion of every possible infection is the first step toward the diagnosis. It is a well-known fact that forms of otitis media purulenta will cause cerebral symptoms and even an increase in the intraventricular fluid, and such otitis is apt to be overlooked, unless thought of at the time a diagnosis is made. The patient, therefore, would run greater danger from such an accident, and would lose a chance of recovery if the diagnosis of otitis or mastoid disease were too long delayed.

Optic neuritis, which is present in most cases, may be present in forms of meningitis of the cerebrospinal type, although Beck puts much stress on this phenomenon. The author is inclined to believe that lumbar puncture will aid more in the diagnosis than any other procedure. The puncture fluid in cases of meningitis serosa thus far published contained no microörganisms, was of low specific gravity, generally 1.007, contained 1 to 1.5 per cent. of albumin, and very few if any cellular elements beyond those of a few blood-corpuscles. On the other hand, a tuberculous meningitis would give a puncture fluid which, though it might in a certain percentage of cases be devoid of microörganisms, would contain a number of mononuclear lymphocytes. In cerebrospinal meningitis the puncture fluid would contain microörganisms unless the meningitis was of a chronic variety, in which form the microörganisms might be absent. In cerebrospinal meningitis, however, a study of the puncture fluid would again aid us, inasmuch as it would show a preponderance of of the polynuclear leucocytes.

XIII. MUMPS.

(*Epidemic Parotitis.*)

Mumps is an infectious and contagious disease of the parotid gland, at times involving the other salivary glands as well as the testis or ovary.

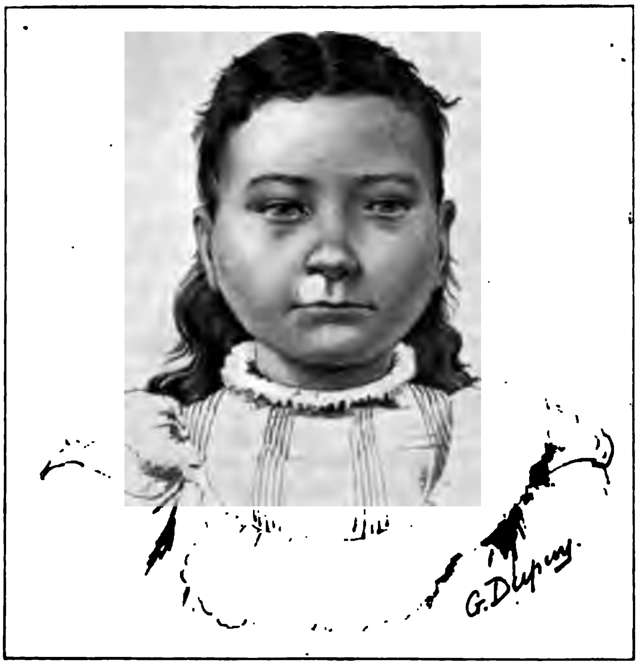
Etiology.—Parotitis is endemic in large cities, and frequently becomes epidemic in schools and institutions where large numbers of children are congregated. It is most common among children of school age, because they are more exposed to infection than children at an earlier or later period of life. Girls and boys are attacked with the same frequency. It may occur in the newly born infant. The author has seen a case in an infant three weeks of age.

The essential cause of mumps is unknown. Laveran and Catlin describe micrococci which they found in the blood and in the glandular lymph of the parotid and testis. These micrococci were arranged in twos and fours, did not stain by the Gram method, and were 1 to 1.5 micromillimetres in diameter. Michaelis and Bein isolated an intracellular chain-forming diplococcus from Steno's duct. The theory thus far advanced is that these micro-organisms gain access to the parotid through the duct. The period of incubation, according to Rilliet and Lombard, may vary from seven to twenty-six days.

Morbid Anatomy.—As the disease is rarely if ever fatal, opportunities to determine the morbid conditions have been few. Virchow first described the condition of the gland as one of inflammatory serous and cellular infiltration of the intra-acinous and peri-acinous connective tissue. The outcome is resolution; induration rarely remains.

Symptoms.—There is a prodromal period, during which the patient is attacked with chilly sensations or a chill, and sometimes with vomiting. There is pain in the region of the ear, and also a ringing in the ears and deafness. There is also a febrile movement, the temperature in some cases mounting to 104° F. (40° C.). The temperature may be normal throughout the disease. There may be headache and loss of appetite. After these symptoms have lasted awhile, the face becomes swollen, as a rule on one side only (Fig. 49).

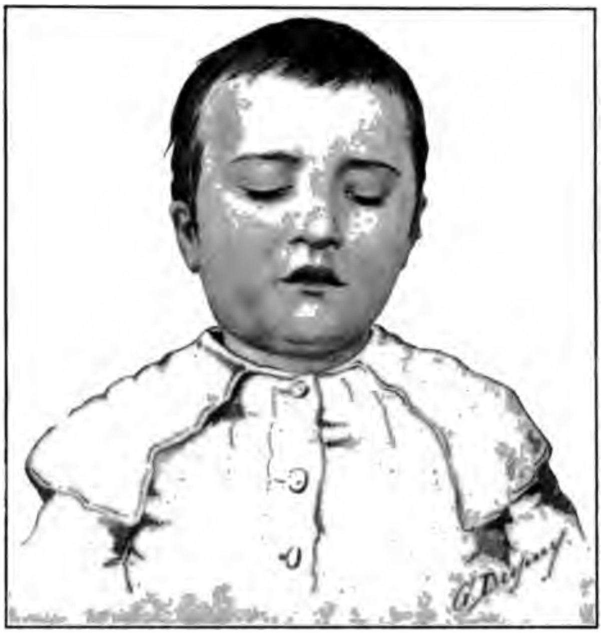
FIG. 49.



Bilateral parotitis.

This swelling gives the face an uneven contour, and is the characteristic symptom. In older children it causes a feeling of tenseness and pain on mastication. Sometimes patients are averse to opening the mouth on account of the pain. In young infants there is drooling. In the majority of cases, after the swelling has lasted three or four days and is subsiding, the opposite side becomes affected. In addition to the swelling of the parotid there is also intumescence of the lymph-nodes of the neck at the angle of the jaw and of the node on the parotid gland in front of the ear. Frequently the submaxillary glands are also swollen, giving the whole face a rounded contour. In most cases the general condition of the patients is good and there is very little discomfort. Other cases have considerable pain and constitutional disturbance. In all my cases there was distinct angina and swelling of the tonsils. In a newly born baby there was swelling of the tissues underneath the jaw and about the larynx, with croupy breathing indicating œdema of the mucous membrane of the larynx.

FIG. 50.



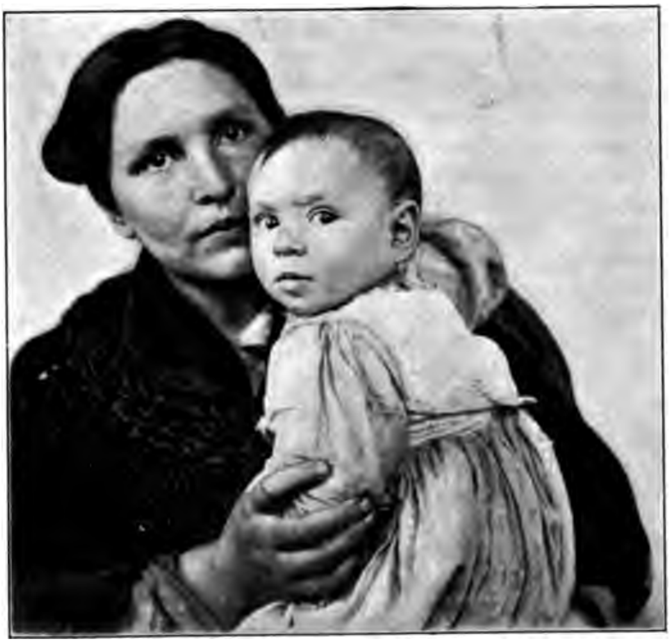
Parotitis involving the submaxillary glands, lateral view. Boy, four years of age.

English authors have described cases in which the submaxillary glands alone were involved, the inflammation being strictly limited to the glands on both sides (Fig. 50). I have seen a few cases of this kind.

Complications.—The testes and epididymis in boys and the ovaries and glands of Bartholini in girls may become affected. There may be ardor urinæ and a urethral discharge. These complications are not so common as the text-books declare. Hydrocele may occur with the orchitis. I have seen a case of this kind in a very young infant. The urine may show a trace of albumin, or in very rare cases there may be blood in the urine. Endocarditis, pericarditis, rheumatism, and osteomyelitis have been reported as complications, but the author has never met such cases. Parotitis complicating pneumonia has been observed in a boy of six years, and in another case otitis and parotitis were present at the same time. In rare cases the breasts and lachrymal glands are affected. Parotitis may be a complication of typhoid fever, measles, varicella, and influenza.

Course.—The disease is at its height in from three to six days, and runs its course in from seven to fourteen days. Mild cases may last only two days. Severe cases are rare. These present cerebral symptoms, and swelling of the tissues about the neck simulating angina Ludovici, with considerable dyspnœa. Cases of recurrent mumps, continuing for from four to six weeks, are recorded. When suppuration occurs, it is probably the result of some mixed infection.

Fig. 51.



Angioma of the parotid simulating mumps.

The **diagnosis** is not difficult. Uncertainty as to whether the parotid is affected or not will be dispelled by drawing a line parallel with the lower border of the jaw; the parotid swelling will be above the line and the lymph-nodes of the neck below it (Fig. 51). In swelling of the mastoid region the ear is raised from the skull, while in parotid swelling, even if it occur behind the ear, that organ remains in its normal position. The swelling of parotitis never fluctuates, but is elastic in character.

The **prognosis** of mumps is good; the majority of cases recover without complications. If the kidneys and endocardium and pericardium are affected, the prognosis will be influenced by the course of these affections. I have never known epidemic parotitis to result fatally.

Treatment.—The patients are isolated and kept in bed as long as symptoms are present. The parotid is anointed twice daily with warm oil of hyoscyamus and covered with cotton. The bowels should be regulated with a saline cathartic. The diet should be assimilable. The affection cannot be controlled by means of drugs. Pain and fever are treated on general principles.

XIV. PERTUSSIS CONVULSIVA.

(*Whooping-cough.*)

Pertussis is an acute specific infectious disease, caused by a micro-organism, probably of the influenza group. It is characterized in the majority of cases by a spasmodic cough accompanied by a so-called whoop.

Pertussis is not only infectious, but it is also contagious. It is propagated through the atmosphere in schools and public places, the air of which is contaminated with the specific agent of the disease. The micro-organism is thought to exist in the sputum and the secretions of the nasal and air-passages of the patient. The disease is especially contagious at the height of the attack. There is reason to believe that the cough of the first or catarrhal stage is highly contagious. The sputum in the stage of decline is also, in my opinion, capable of conveying the disease to others, since it contains the specific micro-organism.

Occurrence.—Pertussis prevails in all countries and climates. It is most frequent during the winter and spring months. It is always endemic in large cities, but, like scarlet fever, becomes at times so prevalent as to be epidemic. Pertussis is essentially a disease of infancy and childhood, but the individual is not exempt at any age. It has been seen in the newly born infant. I have found the disease slightly more frequent in females than in males (1009 out of 1820 cases). Twenty-two cases occurred in infants between one and two months of age. The majority of cases (1343) occurred between the sixth month and the fifth year. The disease is most frequent between the first and the second year (404); next most frequent between the sixth and twelfth month. After the fifth year the frequency diminishes up to the tenth year, after which the disease is very infrequent. Not every one who is exposed contracts the disease. One attack does not necesarily confer immunity, but cases of second attack are rare. It has been observed that pertussis, measles, and influenza frequently follow one another in epidemic form.

Incubation.—The incubation period is variously placed at from two to fourteen days.

Etiology and Bacteriology.—The essential cause of pertussis was believed by Deichler and Kurloff to be a protozoa-like body which they found in the sputum. Afanassjew and Szemetzchenko isolated a bacillus from the sputum. It occurred singly, in pairs or chains, and measured 0.6 to 2.2 micromillimetres in length. The more recent researches on the bacteriology of pertussis are those of Czapelewski, Hensel, and Koplik. Czapelewski and Hensel described in 1897 a non-motile "pole bacterium" or bacillus resembling the influenza bacillus. I at the same time described in the sputum a finely punctate, thin, minute bacillus, 0.8 to 1.7 micromillimetres in length, resembling the influenza bacillus, and staining like that or like the diphtheria bacillus. This bacillus was found recently by Luzatto in cases occurring in an epidemic of pertussis in the city of Graz. It is classified by him as belonging to the influenza group. Positive proof that this bacillus is the cause of pertussis is lacking, since the disease has not as yet been produced experimentally. Evidence simply points toward a bacillus of the influenza group constantly found in the sputum.

Morbid Anatomy.—Post-mortem examination reveals marked inflammation of the nasal passages, bronchopneumonia, and empyema or simple fibrinous or serous pleurisy. Emphysema as a result of rupture of the lung-tissues has been reported by Northrup, who describes the lungs of an infant seven months old as being studded with cavities measuring one-half a centimetre to two centimetres in diameter. The lungs looked like parchment filled with bubbles. Hemorrhages in the eye, ear, and brain are a feature of the morbid anatomy of fatal cases.

Symptoms.—There is undoubtedly a period of incubation, but its length is undetermined, and it can only be said that, if the disease is due to the invasion of a micro-organism, some time must elapse between the invasion and appearance of symptoms. After the appearance of the symptoms there are three stages—the catarrhal, the spasmodic, and the stage of decline. There is no sharp line of demarcation between these stages.

Catarrhal Stage.—This stage in some children is characterized by a cough which is especially troublesome at night, and has sometimes a croupy character. The peculiar nature of the cough becomes apparent when after a few days it becomes more troublesome instead of subsiding. After four or five days it may be accompanied by vomiting once or twice a day, especially if the paroxysm occurs after meals. Examination of the chest may fail to reveal bronchitis. This negative sign is of great value. As the case passes into the spasmodic stage it is noticed that the paroxysms of coughing last longer, and that the child becomes red in the face and expectorates a larger amount of mucus than in ordinary catarrhal conditions. This period of cough without a whoop may last five to twelve days. I have seen many cases in which the whoop was absent in the whole course of the affection. The child had what might be regarded as a severe spasmodic cough followed by vomiting. Fever is present as a rule only during the first few days. It may be remittent and slight. If bronchitis complicates this stage of the disease, there may be a daily rise of one or more degrees in temperature. Usually toward the close of the catarrhal stage the incessant cough causes slight puffiness of the eyelids and slight œdema of the tissues of the face.

The **spasmodic stage** is distinguished by the presence of the characteristic whoop. The cough becomes of a more pronounced spasmodic type. The child has distinct paroxysms, which begin with an inspiration, followed by several expulsive explosive coughs, after which there is a deep, long-drawn inspiration, which is characterized by a loud crowing called the whoop. After one paroxysm has ended, it may be followed by a number of similar ones. When a paroxysm is impending the face assumes an anxious expression, and the child runs to the nearest person or to some article of furniture and grasps it with both hands. The paroxysm is sometimes so severe that the child will fall prostrate or claw the air convulsively. In the severest and most dangerous type a convulsion supervenes. In moderately severe types of the disease the child's face is red or livid, the eyes bulge, and at the end of the paroxysm a quantity of tenacious mucoid or mucopurulent sputum is expectorated. In other cases there is vomiting at the end of the paroxysm. In the intervals the face is livid or pale, or the eyelids are puffy and the face œdematous. In some cases there are punctate hemorrhages on the face, especially about the eyes and temples. There may be chemosis of the conjunctivæ as a result of the bursting of bloodvessels. At this period there is in the majority of cases an accompanying bronchitis, with slight rise of temperature during the day. At first the paroxysms occurring during the twenty-four hours may be few; in some cases they never become frequent, but as a rule they increase in number, so that the patient may have from twenty to one hundred in the twenty-four hours. This stage gradually declines, the number of paroxysms diminishing daily in number and severity. They may subside suddenly or gradually after from four to twelve weeks. The whoop may at times reappear. After the disappearance of the whoop a cough persists for days or even weeks, or it may entirely disappear and suddenly recur with the whoop. It is characteristic of the spasmodic period of the disease that the paroxysms should be more harassing at night than during the day.

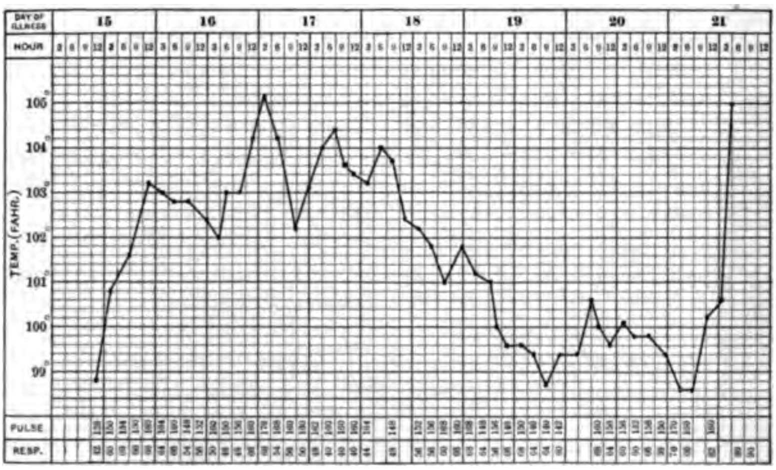
Other Symptoms.—In all cases of pertussis, even in the absence of complications, there is a slight increase in the number of respirations. In cases of even moderate severity the heart impulse is weak, and in exceptional cases the area of superficial cardiac dulness is larger than normal, indicating dilatation of a moderate degree. The pulse is irregular in force and rhythm, and is distinctly more dicrotic than normal. In other words, there is a condition of heart-strain, which is evinced by dyspnœa (even in the absence of exertion), œdema of the face, and cyanosis.

Kidneys.—In the majority of cases a trace of albumin is present in the urine; in others, a few hyaline casts. Blood in the urine is seen in rare cases.

Blood.—Leucocytosis of the polynuclear type is usually present in the second week of the disease.

Complications.—One of the most common complications of pertussis is bronchitis. It may be mild or severe. In the severer form the smaller bronchi are affected, with accompanying bronchopneumonia (Fig. 52). The physical signs are the same as in simple bronchitis and pneumonia without pertussis. In some cases the bronchopneumonia pursues a subacute or persistent course. If reso-

FIG. 52.



Pertussis; disseminated bronchopneumonia in both lungs. Infant eight months of age. Fatal termination.

lution takes place, other areas become consolidated. Emaciation is sometimes extreme. Emphysema is frequently present. Bursting of the air-vesicles may cause pneumothorax, or air may escape into the mediastinum and thence into the neck and into the subcutaneous tissue of the whole trunk.

Hemorrhages.—During a paroxysm there may be epistaxis, conjunctival hemorrhage, bleeding from the ears, and petechiæ on the face and body.

Nervous System.—CONVULSIONS, either general or localized, may complicate pertussis. In the former case the outlook is grave, death usually taking place within twenty-four to forty-eight hours.

PSYCHOSES, such as melancholia and hallucinations, may complicate pertussis. Monoplegia, hemiplegia, or paraplegia, localized facial and oculomotor paralyses, sudden total blindness, deafness, cerebral hemorrhages, hemianæsthesia, and aphasia have been observed.

Gastro-enteritis of a fatal type may ensue.

An attack of pertussis may favor the invasion of the tubercle bacillus. This may have been previously present in the bronchial lymph-nodes or elsewhere in the body, or it may be received into the body during the attack or afterward. In such cases tuberculosis of the lung or other organs, such as the peritoneum, develops.

Diagnosis.—If a cough fails to improve and is especially harassing at night, later in the disease becoming paroxysmal, if the face becomes livid during the paroxysm, if the patient vomits after coughing, pertussis should be suspected and precautions taken to prevent its spread. As a rule, examination of the chest is negative in the first stage. The absence of bronchitis and the presence of a cough of the character described, are characteristic of pertussis. The presence of the whoop dispels all doubt.

Infants who have the incisor teeth and other children may, after the pertussis has lasted for a week, develop an ulceration of the frenum of the tongue, which is called a dentition ulcer. It is caused by friction of the frænum linguæ with the edges of the teeth during the act of coughing. These ulcerations are not diagnostic of the disease; many cases do not show them, and on the other hand they frequently occur in coughs of other forms.

Mortality and Prognosis.—The mortality of pertussis is greatest during the first year of life (25 per cent., Voit). Between the first and the fifth year it is about 5 per cent., and from this time to the tenth year, 1 per cent. (Monti). The occurrence of pneumonia in children under two years of age adds largely to the mortality. Rachitis or marasmus will militate against recovery. Hygienic surroundings render the prognosis more favorable.

Treatment.—**Prophylaxis.**—The patient should be isolated, and should sleep in a large, well-ventilated room. During the day the sleeping-room may be filled for an hour with the vapor of formalin (set free by means of a small formalin lamp). The object is to destroy suspended germs. If two communicating rooms are available, they may be occupied alternately every twenty-four hours, the unoccupied room being fully ventilated in the interval. In this way reinfection may be avoided.

In spring and summer, if the weather is favorable, the children should be constantly in the open air during the day. In large cities the mother is directed to take the child into the park. When in the open air the paroxysms are usually notably lessened. The child should be warmly clad in winter. Sea air seems to aggravate some cases and benefit others. Pine woods and moderately high altitudes are probably the most beneficial, for the patients are not exposed to the unfavorable climatic conditions peculiar to the seacoast.

Medicinal treatment consists of inhalations, topical applications, and internal remedies. Simply to enumerate all the remedies which have been proposed and used in pertussis, would take up the space of a monograph. Inhalation of ozone has been advocated by Caillé. The remedy is expensive and the apparatus not readily procurable. Inhalation of a mixture of 20 per cent. nitrous oxide and 80 per cent. oxygen is beneficial in cases in which the heart is weak. The inhalations are given with a cone for ten minutes twice daily. Insufflation of quinine or other drugs has not proved beneficial. The practice seemed to intensify the paroxysms. Prior, Coggeshall, and others have proposed the application of solutions of cocaine, 4 per cent. to 10 per cent., to the nares and throat. I have had no experience with this method, nor with the local application of antitussin.

If the cough is very troublesome, I first endeavor to control it with full doses of antipyrin combined with tincture of digitalis. The digitalis, in doses of a drop or two several times daily, supports the heart, as is shown by the rapid disappearance of the œdema and cyanosis after its administration. Antipyrin is given in doses of grain j (0.06) for every year of age up to grains v (0.3) every three hours. If the cough is not perceptibly relieved by this remedy after forty-eight hours, I suspend its use, and give codeine in full doses every three hours. Codeine is to be preferred to morphine, which is advocated by Henoch. If vomiting is severe, the food is given in very small quantities in fluid form every few hours. By this method food is retained and absorbed, whereas a full meal is invariably rejected. The use of belladonna has not impressed me favorably. In several cases it seemed to aggravate the cough by causing a dryness of the laryngeal mucous membrane. Bromoform I consider dangerous and of questionable utility. Quinine in full doses three or four times daily is a favorite remedy with many pediatrists. Vaccination and the injection of diphtheria serum have been proposed to abort the disease. I have had no experience with the serum treatment. In a word, the treatment of pertussis consists in applying the rules of hygiene, in mitigating the cough with antipyrin or preferably codeine, and in supporting the heart with digitalis. The complications should be treated on the principles laid down in the sections on Bronchitis, Pneumonia, and Pleurisy.

XV. DIPHTHERIA.

Diphtheria is a contagious febrile disease which affects the throat and air-passages. It is characterized by the formation of a pseudo-membrane on the parts affected. The disease manifests itself by a local lesion and general symptoms caused by the entrance of toxins and, at times, of bacteria into the blood and lymph.

Age and Occurrence.—Although diphtheria is uncommon in the newly born infant, statistics of large numbers of cases show a certain percentage in these subjects; thus, of 547 cases reported by Monti, the newly born number 24, and in Baginsky's statistics several cases are noted. The disease is more frequent from the first to the third month than from the third to the tenth month (Monti). The largest number of cases occur from the second to the sixth year (40 to 63 per cent.) (Monti, Baginsky).

According to Seitz, it is slightly more frequent among boys than girls. Strong as well as weakly children are attacked. Children who suffer from nervous affections, such as poliomyelitis, are more likely to contract the disease than others (Baginsky). All exposed to infection do not contract the disease, because some individuals are immune. Escherich and Fischl have proved that the blood of convalescents contains antitoxic elements. Cases of several attacks in the same individual are not uncommon. Racial peculiarities have no influence.

Diphtheria is prevalent in all parts of the world and epidemics occur at all seasons of the year. It is more common among the poorer classes, not on account of uncleanliness, but as a result of overcrowding.

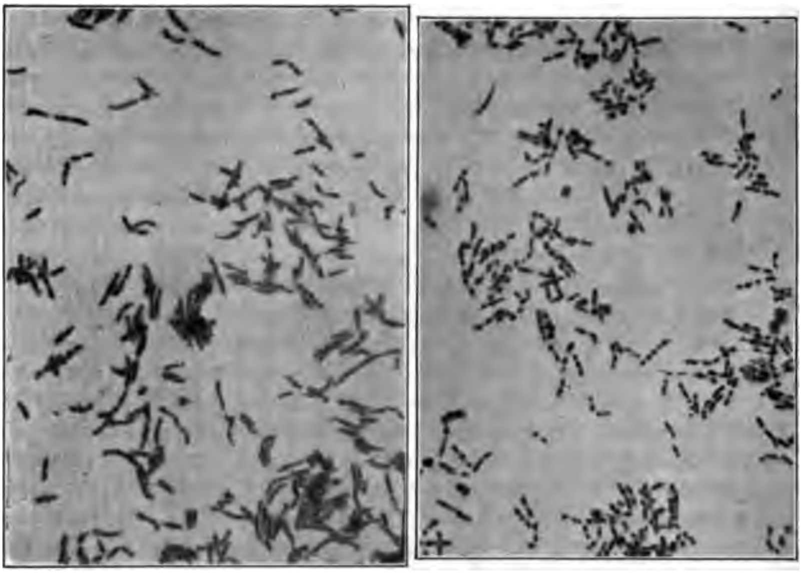
Contagion.—Diphtheria is contagious from person to person, and may be conveyed by any one who has been in the room occupied by a patient with the disease. Mild cases may give rise to fatal cases. The disease is infectious, spreading through families and schools, and may be conveyed through the medium of toys, clothes, and in milk.

Period of Incubation.—This has not been determined with any accuracy in diphtheria. Two to eight days, or an average incubation of three days, is laid down by most observers, but no accurate data are available on this important point. Some authors place twenty days as an extreme limit of incubation. This latter period is evidently only founded on surmise.

Etiology.—The essential cause of diphtheria is a bacillus, the

Bacillus diphtheriæ, which was first noted in stained specimens by Klebs in 1882. Löffler first isolated and accurately described it in 1884. It is present in all cases of true diphtheria of Bretonneau. In the 3 per cent. of cases in which it is reported absent there is good reason to believe that failure to establish its presence was due to imperfect technique. The bacillus is non-motile, twice as thick and about as long as the tubercle bacillus, thickened at the extremities, has no spores, and in some forms has been described as branching. It is very resistant, adheres to clothes and candy, and has been found in milk. It will retain vitality a long time in dried membrane (seventeen weeks), as has been shown by Roux and Yersin. It has been detected nine weeks after the disappearance of the membrane from the throat. It is found present with other bacteria, principally staphylococci and streptococci, pneumococci, Bacillus coli commune, pyocyaneus, proteus, and sprue. It has been found by Roux and Yersin in the throats of perfectly healthy individuals, and may be present without the formation of a membrane. It has been shown that this bacillus forms toxins of very positive action. According to Sidney, the toxins of diphtheria may be divided into albuminoses and organic acids.

FIG. 53. FIG. 54.



The Bacillus diphtheriæ (Klebs-Löffler).

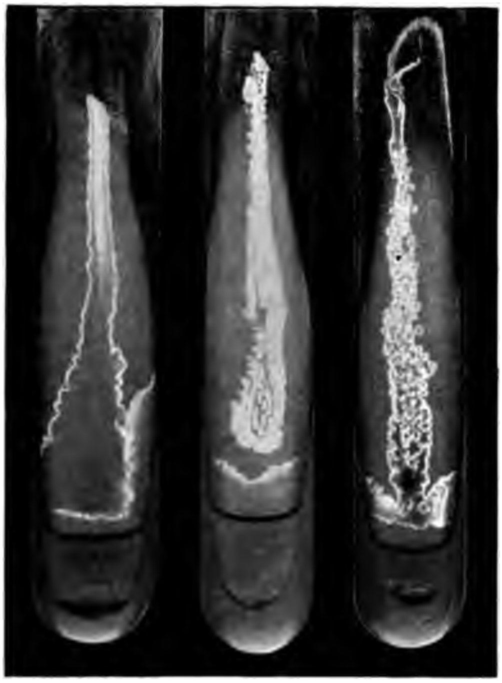
FIG. 53.—Pure culture, photomicrograph. × 1000.

FIG. 54.—Pure culture, photomicrograph. × 1000. Shows the irregular beaded stain.

The pseudobacillus of diphtheria was first isolated by Hoffman. In its growth and staining properties it is identical with the true diphtheria bacillus, but is not virulent to animals. Roux and Yersin regard it as a weakened diphtheria bacillus. Others believe that it bears no relation to the true bacillus. It is found associated with the true bacillus, and also in cases of diphtheria after this bacillus has disappeared from the throat (Koplik). Some authors have given the name pseudodiphtheria bacillus to another variety of bacilli, but this term should be strictly limited to the form described above.

General Infection with the Bacillus Diphtheriæ alone and with Other Bacteria. The bacillus of diphtheria was first demonstrated by Frosch (1895) in the heart's blood, liver, spleen, kidneys, and lymph-nodes. Since then, Kolisko, Paltauf, Schmorl, Booker, Councilman, Mallory, and Wright have demonstrated its presence in the blood and internal organs in fatal cases of diphtheria. The work of Councilman and his pupils is the most recent and complete on this subject. They show that the bacillus may occur alone or in association with streptococci or staphylococci in the blood, lungs, liver, spleen, and kidney. It is more likely to be found alone in fatal cases of uncomplicated diphtheria. The mixed infections with streptococci and other bacteria occur in diseases, such as scarlet fever and measles, which may

FIG. 55.



1 and 3. Cultures of the pseudobacillus of diphtheria on agar, showing the diffuse character of the growth. 2. Growth of Bacillus diphtheriæ (Klebs-Löffler) on the same medium. It is a delicate growth in colonies.

be complicated with diphtheria. The investigators just mentioned found endocarditis, bronchopneumonia, empyema, mastoid disease, and thrombosis of the sinuses due to the diphtheria bacillus. The bacillus was found also in the pus of acute abscesses in various localities.

Morbid Anatomy.—In fatal cases the membrane appears as a thick brownish or grayish-brown mass. It is sometimes present as a thin whitish pellicle, and occasionally is almost black. It may be friable or as resistant as cartilage, and may extend over the tonsils, palate, pharynx, base of tongue, epiglottis, and trachea. The areas not covered by membrane are injected, and may be the seat of hemorrhages. The tonsils are enlarged and bluish red. In the gangrenous forms the tonsils, soft palate, and uvula may be converted into necrotic masses. The nasal passages may show membranous deposit. The epiglottis and vocal cords are thickened. The tracheal mucous membrane is hyperæmic and swollen, there may be adherent membrane, or the pseudomembrane may be loose and curled up in the lumen of the trachea.

The membrane itself has been described by Virchow as croupous and diphtheritic. Councilman is of the opinion that little is to be gained by adhering to the old classification of croupous and diphtheritic membranes. Baginsky also describes forms of diphtheria in which the membrane possessed both croupous and diphtheritic structural characteristics. According to Councilman, the first step in the formation of the membrane is a degeneration and necrosis of epithelium, preceded by a proliferation of the nuclei of the cells. Detritus and hyaline masses result. An inflammatory exudate rich in fibrin is thrown out from the underlying tissue. The fibrin forms in part a reticulum enclosing cells and degenerated epithelium, and in part a hyaline reticulated membrane. The hyaline membrane is formed on surfaces which are covered with several layers of epithelial cells. Fibrinous membrane is formed on the surface and in the tissue. By constant accretions thick masses are formed. The membrane is never formed on an intact epithelium, but may extend over it. There is nothing specific in the diphtheritic membrane. The connective tissue and the bloodvessels beneath the membrane may be the seat of hyaline degeneration. The mucous glands are degenerated.

The diphtheria bacilli are found growing in the necrotic tissue and in the exudation, never in the living tissue or in epithelium undergoing primary degenerative changes. In exceptional cases they may be found enclosed in pus-cells and necrotic epithelium. They are found in masses, and when deeply situated have been covered up by later formation of membrane.

Heart.—Councilman, Mallory, and Pearce have recently described the myocarditis sometimes complicating diphtheria. There is a fatty change in foci or in more diffuse areas in the muscle-fibre. In another form of myocarditis there are interstitial changes, consisting of focal collections of plasma and lymphoid cells, and the formation of new connective tissue, resulting in some cases in a fibrous myocarditis. These pathologic changes are due to the action of the diphtheria toxins on the heart-muscle.

The Lungs.—Councilman states that the most common lesion in fatal cases is a bronchopneumonia, lobar pneumonia never being present. The process begins in an infection of the atria. The bacteria found in the lung, and which are present independently of the character of the lesion, are the pneumococcus (rarely), Streptococcus pyogenes, and the diphtheria bacillus. Marrow-cells are found in the capillaries, and thrombi in the larger vessels. The lymphatics are dilated and contain fibrin and cells.

The **spleen** macroscopically is normal; microscopically, the lymph-nodules are more prominent than is normal, and contain foci of epithelioid cells. The vessels are the seat of hyaline degeneration, and in the later stages contain large numbers of plasma-cells. Some of the nodes may be the seat of necrosis and abscess.

Liver.—The changes in this viscus are due to the action of toxins, and consist of parenchymatous degeneration and necroses, seen especially in the centre of the lobules. There is slight hyaline degeneration of the capillaries.

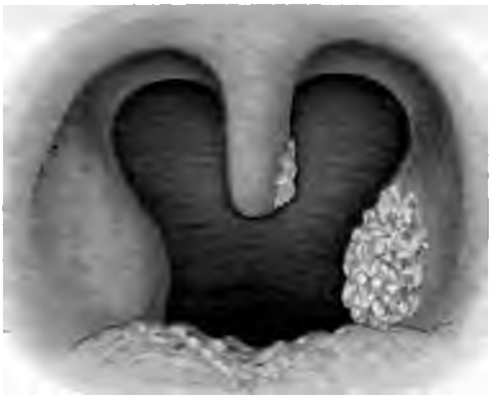
Kidneys.—There may be simple degeneration or acute nephritis. The severe forms of nephritis are found in the cases which are quickly fatal (Councilman). The interstitial and glomerular changes are more common in older children and in protracted cases. There is no specific form of nephritis in diphtheria, and all the changes are due to the action of toxins.

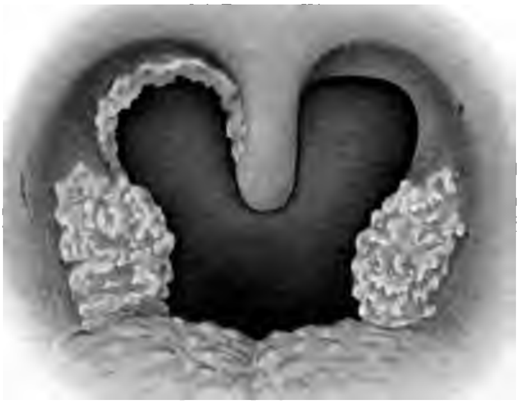
Lymph-nodes.—The mesenteric lymph-nodes, the nodes at the angle of the jaw and in the retropharynx and œsophagus are enlarged, and may undergo necrotic changes (Flexner). Councilman, Mallory, and Pearce describe the changes in the lymph-nodes as being more marked in those nearest the lesion. There are congestion, hemorrhages, and diffuse and circumscribed necrosis. In addition there is a formation of foci resembling miliary tubercles, and composed of epithelioid cells which undergo degeneration, forming granular detritus. Bacteria are not found in the nodes. The changes are due to the toxins.

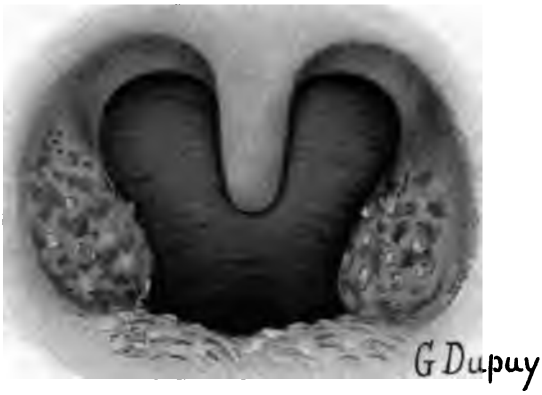
Nerves.—There are fibrillation, increase of the cells of the sheath of Schwann, fatty degeneration of the axis-cylinder, hemorrhages, and nodular degeneration of the nerve-sheaths. In the spine there are infiltration of the meninges, hemorrhages, and degeneration of the anterior horns. Degenerative oculomotor changes are present. There are dilatation and round-cell infiltration around the central canal of the cord.

Stomach.—Diphtheritic membrane in the stomach occurring in cases of diphtheria has been described by Smirnow and Councilman. Of 220 cases reported by the latter, 5 showed the pres-

PLATE XII.







1. Tonsillar Diphtheria, with a small patch of membrane on the uvula.
2. Tonsillar Diphtheria, with a patch of membrane on the pillars of the fauces.
3. Acute Follicular Amygdalitis, which may be diphtheritic.

ence of membrane to a greater or less extent. The membrane either covered the whole surface or formed patches or streaks over the rugæ. The mucous membrane was swollen, hyperæmic, or hemorrhagic.

The Middle Ear.—Of 144 cases reported by Councilman, Mallory, and Pearce, 86 showed involvement of the middle ear on one or both sides; in 7 the mastoid was affected. The inflammatory products were serum or pus. The organism most constantly present was the streptococcus, but the diphtheria bacillus has been found, as have also the staphylococcus and pneumococcus.

The Blood.—The specific gravity is increased at the height of the disease. In mild cases it is not perceptibly changed; in severe septic cases it may range from 1054 to 1060 (Baginsky). Hæmoglobin is reduced only in severe cases of protracted course. Leucocytosis is not marked in mild cases, but in severe septic forms an increase of the white blood-cells has been observed by Felsenthal and Monti. In malignant cases there is a reduction in the number of red blood-cells (Ewing, Billings, Morse).

Symptoms.—Clinically, it is convenient to divide diphtheria into the purely local forms with few constitutional symptoms, the local forms with symptoms of marked toxæmia or septic forms, and the laryngeal forms.

Purely Local Forms with Slight Constitutional Disturbances.—In diphtheria sine membrana, synanche contagiosa (Senator), or catarrhal diphtheria, there may be no formation of membrane, the fauces showing only an angina of varying severity. In some cases there is the picture of a follicular or lacunar amygdalitis. Macroscopically there is nothing to show that the process is diphtheritic (Plate XII). In other forms the membrane is present on the tonsils as specks or strips of exudate, or white or greenish pultaceous masses which may extend to the uvula, or there may be spots or extensive plaques on the posterior pharyngeal wall. In other mild cases the process is confined to a small necrotic excavated area in one or the other tonsil, as described by Henoch. In still other forms the membrane may cover both tonsils, and extend over the soft palate and pillars of the fauces. In these forms of localized diphtheria the nares are seldom involved.

In these localized forms of diphtheria the infant or child may present few symptoms pointing to the throat affection. Unless the physician be systematic in his methods of examination, he may fail to inspect the throat at his first visit, and the diphtheria may thus escape detection. The nursling in this as in the non-diphtheritic affection, may refuse to take the breast. The movements are greenish, and have an offensive odor, or may be diarrhœal. There are fever and restlessness. Inspection will reveal slight or marked swelling of the lymph-nodes at the angle of the jaw. The temperature may not be above 101° F. (38.3° C.) or may be as high as 105° F. (40.5° C.). As a rule, it is not persistently high. The pulse is accelerated and the respirations slightly increased. The invasion of the disease is for the most part insidious in nurslings; rarely is there a chill or convulsion. The tonsils are enlarged, and show small specks or plaques of membrane on their surface. The uvula may be red and swollen, and there may be patches of membrane on the sides adjacent to the tonsils. There is sometimes a croupy cough. In purely local diphtheria, however, the larynx is not involved in the majority of cases. The urine may show a trace of albumin, and in some cases a few leucocytes, blood-cells, and a very few hyaline casts. In older children the signs of illness are more marked. They complain of pain on swallowing, and the temperature may at first be high. Toxæmic symptoms, such as pain in the joints, headache, pain in the back, and slight prostration, are present. Inspection of the throat may show the tonsils to be enlarged, and to present the appearances mentioned above. Other members of the family may complain of sore throat. I have reported cases in which children complained of but few symptoms and engaged in their customary play. Examination of their throats disclosed the presence of simple inflammatory redness and swelling of the tonsil, pharynx, and uvula. In these cases the diphtheria bacillus was detected in scrapings from the fauces. Membrane never developed, and yet they were cases of true diphtheria.

The fever is not characteristic. The temperature may at first reach 104° F. (40° C.) or above, and gradually drops to the normal with subsidence of the symptoms. Otitis and suppuration of the submaxillary and retropharyngeal lymph-nodes may cause the temperature to become remittent or intermittent.

Septic Form of Diphtheria.—In the second clinical form of diphtheria there are, in addition to the local symptoms present in the first form, constitutional symptoms of a severe or even septic type. The children at the outset appear very ill; the temperature is high, there is marked restlessness with a tendency to drowsiness, the face is flushed, and the breathing noisy or nasal. The infants refuse the breast or bottle, and older children complain of great pain in swallowing. In some cases the glands at the angle of the jaw are swollen, and the neck is more rotund than normal. Inspection of the throat shows the membrane on the tonsils, or on both uvula and tonsils. It spreads rapidly, the tonsils, soft palate, and pharynx being covered in one or two days. The membrane may break down, and masses of necrotic tissue be expectorated. In severer forms the membrane extends over the posterior nares, and gradually invades the nasal passages. At first a slight nasal serous discharge is noticed, which increases in amount and becomes ichorous and tinged with blood; the anterior nares become eroded and are coated with a whitish or greenish membrane. In some cases the membrane involves the buccal mucous membrane. There is severe stomatitis, the lips are eroded, and the angles of the mouth may show rhagades covered with membrane. With the development of these symptoms the toxæmia increases; the fever may be moderate, not exceeding 102° or 103° F. (38.8° or 39.4° C.); the pulse is rapid and feeble; the sensorium somewhat benumbed. The lymph-nodes at the angle of the jaw may be much enlarged, and the tissue underneath the jaw may be the seat of phlegmonous inflammation. The breath has a very fetid odor. The urine may reveal the presence of albumin, a slight amount of blood, and a few casts of the hyaline or epithelial type.

The constitutional symptoms may diminish in severity, and with the subsidence of the local symptoms the appetite returns, the sensorium brightens, and recovery gradually takes place. On the other hand, if a fatal issue occurs, it results from heart paralysis, paralysis of the general nervous system and respiratory function, or extension of the diphtheritic process to the larynx, trachea, and lungs.

If the diphtheria extends to the larynx, the voice becomes first husky, then croupy. The breathing is labored and of the laryngeal or croupy type, there is retraction of the suprasternal notch and epigastrium, the accessory muscles of respiration are drawn into play, and unless relieved the patient dies of suffocation. Even if relieved, when the septic symptoms and toxæmia are severe the patient may succumb or the process may spread downward, and involve the trachea and lungs. In those cases in which there is cardiac paralysis, vomiting and abdominal pain supervene. The patient is pale and the surface cool. Gallop rhythm sets in and the heart-sounds become indistinct. The expression is at first anxious, then apathetic; the voice is scarcely audible; the patients no longer notice their surroundings. Death ensues from pulmonary œdema with symptoms of heart-failure.

If the general nervous system is involved, paralysis of the soft palate sets in even after the membrane has disappeared from the tonsils and pharynx. The reflexes are absent, and the child is unable to sit upright. The act of swallowing not only becomes difficult, but fluids may find their way into the larynx and thence into the trachea, causing pneumonia; or the paralysis may extend to the diaphragm, when the lethal issue is hastened by paralysis of the respiratory apparatus.

The Malignant Septic Form.—This form has been partly described above. It is characterized not only by the malignancy of the local process, but by the severity of the toxæmic symptoms as well. It was formerly believed that these cases were due to mixed infections with streptococci and staphylococci, but it is now known that the

Bacillus diphtheriæ alone may cause all the symptoms. In these cases not only the toxins, but the bacillus itself also enters the circulation. The pharynx, tonsils, and nares are covered with a dirty brown or greenish membranous exudate. There is an ichorous discharge from the nares. The tonsils, pharynx, and lymph-nodes of the neck become necrotic. The membrane is discharged from the nose and mouth. The fetor of the breath is extreme, and the prostration correspondingly great. The larynx, trachea, and lungs may be involved in the diphtheritic process. The pulse is weak and rapid. The temperature may not be above the normal, and in some cases may be subnormal. Acute nephritis may be present. In some cases hemorrhage under the skin and from the nose, mouth, bowel, and even kidney, may precede death.

A few cases recover, but in them the necrosis of tissue in the pharynx and larynx causes permanent defects and cicatricial contractures. Loss of the uvula and perforations of the soft palate may result from diphtheria in early life.

Laryngeal diphtheria (*croup*) is the result of the extension of a mild or severe tonsillar or pharyngeal diphtheria. There may be no preceding clinical manifestations. There are the rare cases of so-called ascending croup, whose existence has not been wholly disproved. Cases are seen in which the most careful inspection has failed to detect preceding disease of the pharynx, epiglottis, or tonsils. Lastly, there is a class of cases which occurs during convalescence from pharyngeal or tonsillar diphtheria.

The symptoms vary accordingly as the disease manifests itself first in the larynx or follows a localized tonsillar or pharyngeal diphtheria. In the latter case there may be slight redness of the tonsils or pharyngeal mucous membrane, or the parts above the larynx may show membranous deposits. In either case the laryngeal invasion is ushered in by croupy cough and stridulous or metallic breathing. The cough is harassing and persistent, and the stridor increases within twenty-four or forty-eight hours to such an extent as to be distinctly audible, and to give the impression that there is a mechanical obstruction in the larynx. The breathing becomes labored, and there is retraction of the parts above the sternum and of the peripneumonic groove, especially at the epigastrium. In rachitic infants the sides of the chest and the epigastrium are markedly retracted at each descent of the diaphragm. With increasing obstruction the face assumes an anxious expression, the lips become cyanosed, and the surface cool. The pulse is rapid—120 to 180. The fever may be high or low. The lividity of the face in the severer forms of dyspnœa gives place to pallor. The picture of laryngeal obstruction, with the stridulous breathing, increased respirations, and overaction of the accessory muscles of respiration, is so characteristic as to be significant to even the inexperienced observer. During the paroxysms of coughing membranous casts are expelled from the larynx. The membrane may extend downward, involving the trachea and bronchi, casts of which may be expelled. The lungs may become involved, and in severe cases are the seat of a bronchopneumonia of streptococcic nature. With this there may be compensatory emphysema. The urine may show the existence of slight or extensive nephritis, or may be normal in every respect.

Especially deceptive are those cases of membranous laryngeal diphtheria or croup whose onset closely resembles that of so-called catarrhal laryngitis. In these the symptoms may develop suddenly, and within twenty-four hours the patient presents all the symptoms of laryngeal obstruction (croup d'emblée of the French). Inspection may show little variation from the normal appearances in the pharynx. We should be cautious not to assume that no membrane is present in the larynx. Cases have been recorded in which laryngoscopic examination failed to show membrane in the larynx, but in which post mortem it was found present beneath the cords and in the trachea.

Course and Duration.—In the mildest and purely local forms the disease reaches its height in from two to four days; the temperature then drops to the normal and convalescence is established. In the severe septic forms the membrane spreads from the tonsils to the pharynx, and the disease attains its full development in from five to eight days. The temperature falls by lysis or crisis, and convalescence is established. If the case is very severe, the disease shows no tendency to limit itself, the toxæmia is extreme and the involvement of the lymph-nodes is very great. Death may ensue in from a week to fourteen days. In some very malignant cases death may ensue in from three to four days after the onset of the disease. The laryngeal diphtheritic croup reaches its full development as a rule early—within three days. The disease may then retrograde under treatment or may advance into the trachea and bronchi, and cause death in a variable length of time.

The **complications** include bronchopneumonia, pleuritis, gastroenteritis, retropharyngeal abscess, suppuration or necrotic destruction of the lymph-nodes of the neck, nephritis, cardiac paralysis, early and late (or post-diphtheritic) general paralysis, and diphtheria of the eyes, skin, and vulva.

Bronchopneumonia and Pleuritis.—Bronchopneumonia is found in from 50 per cent. (Baginsky) to 80 per cent. (Talamon) of the autopsies on children who have died of diphtheria. It results from extension of the disease from the trachea into the smaller bronchi and alveoli of the lung, and is therefore always a true bronchopneumonia. Through the investigations of Löffler, Flexner, Northrup, and Prudden, it has been proved that the diphtheria bacillus, the Streptococcus pyogenes, the Staphylococcus pyogenes, and the pneumococcus are the exciting causes of the pneumonia. In the pneumonia resulting from the diphtheritic or pseudodiphtheritic processes complicating scarlet fever and measles, Prudden and Northrup have shown that the Streptococcus pyogenes is an active causal agent. The onset of a complicating pneumonia is generally indicated by an exacerbation of the dyspnœa, fever, and cough. The prostration is also more marked. Auscultation of the inferior lateral or posterior parts of the chest on one or both sides reveals the presence of bronchopneumonia; while resolution is taking place in one part of the lung, other areas are being involved. Thus an apparent improvement may be followed by a rapid rise of temperature, increased dyspnœa, and rapid pulse. This form of bronchopneumonia may be complicated by pleuritis of a serous, serofibrinous, purulent, or hemorrhagic type.

Gastro-enteritis.—In nurslings there is frequently a diarrhœa with green stools and vomiting. In some cases these symptoms may become severe. Extension of the membrane into the œsophagus, stomach, and gut may take place, with a fatal result. The cases of simple diarrhœa are directly due to the swallowing of bacteria from the mouth and fauces. The diarrhœa may be so severe as to become one of the leading features of the disease.

Retropharyngeal abscess occurs in the tonsillar and pharyngeal forms of diphtheria as a result of infection of the retropharyngeal lymph-nodes by streptococci.

Nephritis may be absent, slight, or severe. Baginsky found it present in 42 per cent. of his cases. In the majority of cases of even mild diphtheria there is albuminuria; in some the urine may, in addition, contain casts, blood-cells, renal epithelium, and leucocytes, showing grave lesions of the kidneys.

The affection of the kidneys is brought about by the action of the toxins on the parenchyma of the kidney. Not only are toxins produced in the kidney substance, but bacilli have been found in the kidney and in the urine. A large percentage of the cases of nephritis are of the mild type. Here, as in scarlet fever, we have cases in which there is nephritis with blood-casts and uræmic symptoms in the course of the disease, and cases in which there is total suppression. All are agreed that œdema and anasarca of the body are uncommon, even in the presence of severe nephritis. I have seen severe septic forms of pharyngeal diphtheria ushered in with vomiting and uræmic symptoms, such as headache and exhaustion, before the appearance of the membrane. These symptoms subsided when the membrane was fully formed, to be followed in a few days by complete suppression of urine after the disappearance of the membrane. In one of my cases the membrane had entirely disappeared from the throat and the patient was apparently convalescing when total suppression set in, continued for several days, followed by uræmic convulsions and death.

Heart Paralysis.—Of greatest clinical significance is the cardiac diphtheritic paralysis, which may become apparent either early during the course of the disease or later on in convalescence. The early form may set in while the membrane is still visible in the throat. It occurs in the septic forms of the disease. These are the severe cases. The children show great prostration and apathy; the pulse is rapid and irregular; the heart-sounds, especially the muscular sound, is indistinct; the pulse is feeble and flickering; there are vomiting and abdominal pain. These symptoms may repeat themselves in attacks, until finally the patient dies with all the symptoms of collapse, such as cool extremities and shallow respirations. In such cases there is, as a rule, a marked nephritis. In the late cases the symptoms of cardiac failure appear from the second week of the disease to the seventh week of the convalescence. The membrane has disappeared from the throat. There may be no premonitory symptoms, or there may have been a slight blowing murmur at the apex. In their mildest form the heart symptoms appear in the second or third week. The heart becomes irregular, and the muscular sound is weak; the pulse becomes small and either slow or rapid (tachycardia). There may be attacks of syncope, during which the patients vomit, complain of abdominal pain, and refuse medicine and nourishment. Sudden cardiac failure and death without symptoms, premonitory or otherwise, may occur in the period of convalescence.

Mild forms of cardiac irregularity which do not eventually prove fatal are seen in the beginning of convalescence. There are forms of cardiac irregularity which may appear alarming at first and in which complete recovery results. Thus, as will be seen under the heading of Myocarditis, it is not uncommon in the convalescence, early or late, to observe the heart become irregular. This irregularity increases from day to day. In its most pronounced form I have observed it in a child three years of age, in whom the heart would contract two or three times, there would then be a pause, followed by two or three or four contractions. The pulse varied from 80 to 96 during sleep, and 110 to 130 in the waking state. The compressibility of the pulse varies in these cases; the heart-beat is weak, or at times may be strong. The second sound will be at the pulmonary orifice. In these cases the child is apparently comfortable. There is no pericardial distress, pain, or vomiting; there may be occasional sighing. The cardiac irregularity may persist for days, even weeks, and ultimate recovery result. It is not always in the severe cases of diphtheria that these symptoms of cardiac disturbance appear, but in the apparently mild cases of short duration. The severe forms of cardiac paralysis set in with symptoms of the early cases. These symptoms may have been preceded by the milder symptoms of cardiac irregularity. There is slight albuminuria. Suddenly, while in apparent good health, the patients complain of dyspnœa and pain in the stomach. The lips become cyanosed and the extremities cool, the pulse thready, the heart impulse weak, the heart-sounds scarcely audible; the heart may be rapid or as slow as 40 to 50 beats per minute. Vomiting is repeated, and in some cases the liver is enlarged, as also the spleen. I find in all cases of diphtheritic myocarditis that the enlargement of the liver and spleen with the increase of the pulse rate is a symptom of very serious moment, and, as a rule, a precursor of a fatal issue. The patients may survive one or two such attacks, only to succumb finally. In the early forms of cardiac paralysis there may be no gross lesions in the heart-muscle. In the later forms the lesions are more apparent. There are fatty parenchymatous changes. In other cases there may in addition be changes in the vagi.

Diphtheritic Paralyses.—Paralyses are the result of the action of the toxins of the Bacillus diphtheriæ on the nerve-trunks and tissues of the general nervous system. The paralysis may occur in the course of the disease or during convalescence. When the paralysis occurs early, it affects the velum pendulum palati. In cases which result fatally the heart becomes affected, pneumonia caused by the passage of food into the larynx develops, or the paralysis may become general. In the latter case the symptoms are similar to those seen in the post-diphtheritic forms of paralysis. This form of paralysis manifests itself from the second to the sixth week after the onset of the disease. In mild forms, it may begin with a paralysis of the muscles of the soft palate, which remains localized. The child has a nasal tone of voice, and liquid food is regurgitated through the nose on swallowing. In severe cases there are in addition loss of the patellar reflexes, ataxic conditions, inability to sit upright or to stand, oculomotor paralysis, facial paralysis, pallor, weak heart, arhythmia, loss of appetite, and albuminuria.

Recovery may take place even when there is general involvement of the muscles. The great danger is extension of the paralysis to the diaphragm. Post-diphtheritic paralysis occurs in 5 to 7 per cent. of the cases of diphtheria, according to Baginsky, who reported 131 cases of paralysis in 2300 cases of diphtheria. The soft palate was most often affected. Among the other forms of paralysis are those of the facial and oculomotor nerves, the larynx (recurrent laryngeal), and lastly forms of ataxia. Antitoxin has little effect in preventing these paralyses. They occur as frequently after its administration as during the pre-antitoxin period.

In the American Pediatric Society's tabulation 9.7 per cent. of the cases had paralysis; of these, 32 out of a total of 328 cases died of cardiac paralysis.

Hemiplegic cerebral palsy may occur in diphtheria (Monti, Levi, Baginsky).

Disturbances of the sensory nerves also occur in diphtheria, such as perversions of the senses of smell and taste; also anæsthesia of the rectum.

Psychical derangements such as melancholia have been reported.

Diphtheritic Ophthalmia.—True diphtheritic ophthalmia occurs both as an accompaniment of diphtheria of the fauces and as an idiopathic affection. There are two distinct forms of pseudomembranous affection of the eye. In the first, the Löffler bacillus is present, but in the second, or diphtheroid form, it is absent, and the streptococcus alone is found. Of the true diphtheritic form, one class of cases has a mild clinical course. In these the bacillus isolated resembles the pseudodiphtheria bacillus in not possessing virulent properties. In the other form of diphtheritic eye affection the membrane spreads rapidly and causes destruction of the eye. The diphtheritic invasion is ushered in with redness and chemosis. The membrane appears first on the palpebral conjunctiva, and causes marked swelling of the lids. There is little seropurulent discharge. In the progressive form destruction and perforation of the cornea result. I have seen several cases in connection with fatal diphtheria complicating measles, and also cases in which there was no history of diphtheria in the patient or family. I have seen it occur as an idiopathic affection in nurslings. According to Baginsky, diphtheritic ophthalmia occurs in 3 per cent. of the cases of diphtheria, and is most frequent from the second to the sixth year.

Diphtheria of the skin occurs when the specific bacillus finds lodgement in an abrasion or cut. The membrane spreads over the wound and encroaches on the surrounding skin.

Diphtheria of the vulva is met with both as an idiopathic affection and as a complication of true diphtheria elsewhere in the body. I have not found the Klebs-Löffler bacillus in a number of pseudomembranous inflammations of the vulva and vagina in infants. Some of these cases show the presence of true membrane; others begin as aphthous ulceration and develop membrane later. These cases are benign. The diphtheritic bacillary cases may be divided into two distinct classes according to their causation. The cases of one class show the Löffler bacillus, but are benign in course, although I have proved by animal experiment the presence of the bacillus of diphtheria in virulent form. In the other class of cases there is extensive destruction of tissue, and sometimes a fatal result. Cases of this class occur as a complication of diphtheria elsewhere in the body or in connection with the exanthemata.

The symptoms of diphtheria of the vulva and vagina may be localized strictly to the parts, or there may, as in the severer forms of Henoch, be constitutional symptoms of toxæmia. Locally, the disease is characterized by the appearance of patches of membrane on the inner surface of the labia, clitoris, and introitus vaginæ. The parts, especially the labia majora, are intensely swollen and œdematous. In Henoch's cases there was gangrene or necrosis of neighboring tissues. In my cases there was no complicating diphtheria of other parts. The cases occurred in infants and in children under two years. They were benign in course, although of bacillary type.

Nasal Passages.—Councilman, Mallory, and Pearce, in their latest monographs on diphtheria, call attention to the frequency of invasion of the accessory sinuses of the nose and antrum by the diphtheritic process. They found the antrum affected in 33 cases of 52 examined. Clinically, this affection is more common than appears from these figures. This would account, according to these authors, for the persistence with which diphtheria bacilli continue in the nasal secretions after the throat lesions have disappeared. The disease of the antrum may, as pointed out by Wolff, and recently by Mayer, persist after the diphtheria has run its course. Mayer classifies the symptoms as eversion of the lower lid, fistulous opening in the cheek from which pus exudes, and a fetid purulent discharge from the nose on the side of the face at which the fistula is situated.

Other Complications.—Diphtheria in *pertussis* is a serious complication, since the resistance of the patient is generally much decreased. *Bronchopneumonia* is especially to be feared. In *tuberculosis* the patient usually dies as a direct result of the complication. In *measles* the diphtheritic process is a grave complication; it may invade the larynx and death may ensue from extension of the disease to the lungs. In *typhoid fever* the process causes death by invasion of the lungs.

Exanthem.—Is there an exanthem characteristic of diphtheria? I am inclined to view all eruptions which may occur in the course of this disease as purely accidental. They may be the result of remedies (antitoxin) administered or of some infection originating in the gut. Among these eruptions are the various forms of erythema and roseola. Erythema urticatum is often seen.

The **diagnosis** of diphtheria must be considered in its clinical and bacteriological aspects. Clinically the characteristic and ever-present lesion is the membrane. This is seen on the tonsils, uvula, pillars of the fauces, and the posterior pharyngeal wall. Its color varies. In consistency it may vary from a thin pellicle or cloudy discoloration to a thick adherent, pultaceous or stringy mass. In a large proportion of cases the presence of the membrane and other characteristics are presumptive evidence of diphtheria. On the other hand, there are certain forms (not very frequent) of pseudomembranous inflammation of the tonsils and fauces which are not truly diphtheritic; these are called pseudodiphtheria or diphtheroid. In these cases the Klebs-Löffler bacillus is not found, but streptococci, staphylococci, and other bacteria are present. Some forms of diphtheria show at first only fibrinous specks on the tonsils; in others there are small necrotic ulcerations on the tonsil, and in still others the diphtheria may simulate an acute catarrhal follicular amygdalitis or lacunar amygdalitis. These cases are not as infrequent as was formerly supposed. In the pseudomembranous and other forms of inflammation of the throat above described a bacteriological test should always be made. It should be practised as a routine procedure in all cases of angina. Cultures should be made in cases of laryngeal inflammation in which no membrane is visible in the fauces. If membrane be present in the fauces, and a culture fail to reveal the Klebs-Löffler bacillus, a second and even a third culture should be made. I have frequently established the presence of the specific bacillus in membrane in cases in which the first culture-test proved negative. It is not a reliable nor satisfactory method to spread membrane or secretion from the throat direct on a cover-glass, and decide from such a preparation the nature of the process. The technique of culture-tests is scarcely within the scope of this work. It is sufficient to state that growth can be obtained within four or five hours if the culture-tube is subjected to a temperature of 100.4° to 102.2° F. (38° to 39° C.) in a small incubator. Other diseases, such as membranous forms of stomatitis, may simulate diphtheria. In these cases the culture test is the only positive mode of making a diagnosis. Certain forms of laryngismus stridulus resemble acute diphtheritic laryngitis, or a diphtheritic process may be present in the larynx in a rachitic infant subject to attacks of laryngismus. Cultures should be made in all such cases.

In small towns and country districts the practitioner without the aid afforded by laboratories will often be thrown on his own resources in making a diagnosis. In such cases the following clinical symptoms may be considered fairly presumptive evidence of diphtheria:

The presence of membrane on a tonsil and a small patch, streak, or speck of membrane on the adjacent surface of the uvula or tip of the uvula; a patch of membrane on the tonsil and an accompanying patch on the posterior pharyngeal wall; the presence of a croupy cough and stridulous breathing with small patches of membrane on the tonsil or epiglottis, are all of much diagnostic value. The presence of albumin in the urine is of little value in making a diagnosis, as it may be present in non-diphtheritic affections and absent in diphtheria. Constitutional symptoms are only of corroborative value. It is well known that the most virulent forms of diphtheria may at first be manifested by few constitutional symptoms. The temperature-curve is not characteristic. If a patient who at first suffers from a catarrhal tonsillitis or pharyngitis, shows within twenty-four hours minute patches of membrane either on the uvula or pharynx, it may reasonably be assumed that true diphtheria is present. An acute laryngeal inflammation, croupy cough, and stridulous breathing which not only persist beyond the first twenty-four hours or first night, but also become aggravated, justify a diagnosis of diphtheria of the larynx, although no membrane is visible in the throat. General symptoms are of little diagnostic value. Rhinitis at first accompanied by a serous and later by a fetid sanguinolent discharge, with glandular swellings in the neck, is diagnostic of diphtheria.

Adenitis is frequently absent at the outset of tonsillar diphtheria, even when patches of membrane of some size are present. On the other hand, a simple catarrhal tonsillitis is often accompanied by marked adenitis.

Paralysis of the soft palate, appearing in the course of a severe or mild pseudomembranous tonsillar, pharyngeal, or laryngeal inflammation, or after the affection has run its course, points strongly to true diphtheria, although cases of paralysis of the soft palate following diphtheroid have been reported. The color of the membrane, its detachability, and the fact that a bleeding surface is left after its removal, cannot be relied upon as aids to diagnosis, in view of the fact that interference with the membrane is not advisable.

Aphthæ with pseudomembrane over the vault of the hard palate, spreading to the gums and cheeks, are seen in newly born and older infants. These forms of pseudomembranous stomatitis are the result of traumatism inflicted by the infected fingers of the nurse or mother, and are limited to the parts on which they are first seen. Such septic membranes rarely spread unless the exciting causes are perpetuated.

Herpes of the pillars of the fauces, so-called herpes of the tonsils, are often mistaken for diphtheritic patches. With a suitable light such an error should seldom be made.

Following the ingestion of caustic alkali or the traumatism consequent on washing or rubbing the mucous membrane, aphthous ulcerations, which closely simulate diphtheritic membranous patches, are prone to appear over the hamular process of the palate bone. The history of the case, the absence of diphtheria elsewhere, and the result of a culture test will exclude diphtheria.

The patches of necrotic tissue seen on the tonsils, pillars of the fauces, and uvula following tonsillotomy and ablation of adenoids, and sometimes accompanied with paralysis, may mislead the observer and cause him to make a diagnosis of true diphtheria.

The membranous patches which appear on the tonsils of scarlet fever patients at the outset of the disease are for the most part diphtheroid. Unless the patient has been exposed to a double infection, which is infrequent in private practice, the patches of membrane which appear later in the disease are also of a diphtheroid nature. True diphtheria may coexist with scarlet fever (Baginsky, Escherich, Councilman), but does so in only a small number of cases.

The appearance of a pseudomembranous exudate on the tonsils of a patient attacked with measles should be regarded as diphtheritic until the contrary has been proved. The laryngitis with croupy cough and breathing often complicating measles is not, as a rule, diphtheritic.

The **prognosis** and mortality vary with the age of the patient, the form and severity of the infection, and the extent to which organs other than the fauces and larynx are involved. Young infants, unless they come under observation early, give a high mortiality rate. Septic forms of diphtheria are more fatal than those in which the process is a distinctly local affection. The mortality also varies with the nature of the epidemic. In Baginsky's statistics of 2711 cases, the mortality from the sixth to the twelfth month was 52 per cent.; from the second to the third year, 37 per cent., decreasing to 8 per cent. in the tenth year. The death-rate is high in infants and children of delicate constitution and in those suffering from any form of dyscrasia.

The **treatment** of diphtheria may be prophylactic, constitutional, and local.

Prophylaxis.—The patient should be isolated as soon as the membranous deposit is detected. Other children of the family who have been in contact with the patient should at once be given immunizing doses of antitoxin, and the furniture of the sick-room, such as hangings and carpets, should be removed, only the most necessary articles being retained. The room should be well ventilated. The nurse should not come in contact with other members of the family. All articles of clothing worn by the patient should be dipped in an antiseptic solution (corrosive sublimate, 1 : 2000) before removal from the sick-room. The physician, before entering the sick-room, should cover his head with a cap and wear a long coat or bath-robe, which should be hung outside the sick-room. If it is necessary for members of the family to enter the room, they should observe the same precautions, and on leaving the room they should gargle or rinse the mouth with some mild cleansing solution, preferably of boric acid. A throat culture should at once be made. The swab should be rubbed over the tonsils if they are the seat of exudate; if the case is laryngeal, the swab is passed over the epiglottis and posterior pharyngeal wall. Utensils used in feeding the patient should not be used by others.

The patient after convalescence should not mingle with other children until culture has proved the absence of the Bacillus diphtheriæ from the throat.

Constitutional treatment consists first in the administration of diphtheria antitoxin. It is not within the scope of this work to enter into the details of the theory of action of this agent, which is the outcome of the modern experimental method of the investigation of disease. Its place in the therapy of diphtheria is now assured. The mortality of diphtheria has been greatly reduced since its introduction. Baginsky gives the following figures, showing the mortality before and after the introduction of antitoxin:

Age.	Before.	After.
Two years	60.2 per cent.;	25.8 per cent.
Two to four years	51.2 " " ;	17.1 " "
Eight to ten years	28.8 " " ;	10 " "

Of 5794 cases in private practice collected by the American Pediatric Society, the total mortality was only 12.3 per cent. In the cases injected on the first day of the disease the mortality was 7.3 per cent. In the laryngeal form of diphtheria the results have been especially favorable. In 1704 cases operated and not operated there was a mortality of 21 per cent., of the intubated cases, 23 to 27 per cent., as against 60 to 70 per cent. before the introduction of antitoxin.

The DOSAGE varies with the age of the patient, the severity of the infection, and the duration of the case before the beginning of treatment. Mild forms of local membranous affections of the tonsils and pharynx coming under observation on the first day should receive doses of antitoxin as follows: Up to one year, 600 units; one to two years, 1000 units; two to five years, 1500–2000 units. If the disease has markedly progressed twenty-four hours after the first injection, the initial dose should be repeated. The severer forms of localized diphtheria with marked constitutional symptoms should receive initial doses half as large again or twice as large. Laryngeal forms should receive proportionately large doses. The American Pediatric Society recommends as an initial dose 1500 units for a child under two years, and 2000 units for one above that age. I employ 300 units for immunizing purposes in very young infants, and 500 units in older children.

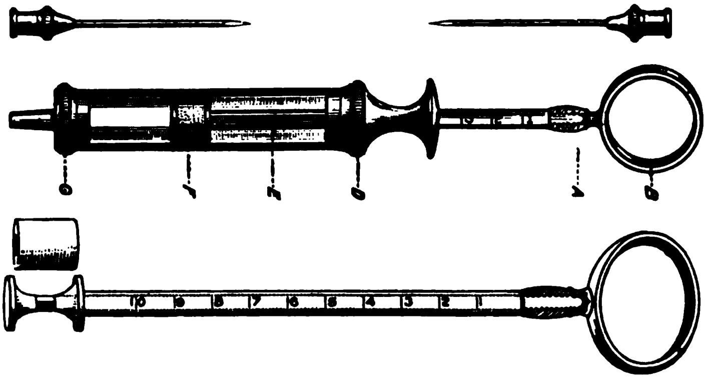
The immunizing power extends over a period of three weeks. It is best to give an initial dose of sufficient amount, so that a repetition of the dose will not be necessary; on the other hand, it is advisable not to give an excessively large dose. The concentrated antitoxins are preferable both on account of the diminished bulk and the infrequency with which skin and joint-affections follow their injection. Recently prepared antitoxin should be obtained, for it has been shown that this agent deteriorates with age (Abbott), and then no longer contains the original unit values.

TIME OF INJECTION.—The antitoxin should be given as early in the course of the disease as possible. If membrane is present, no time should be lost in waiting for the result of the culture test, for if the disease is not true bacillary diphtheria no harm can result from the injection, while to wait may be hazardous to the patient.

MODE OF INJECTION.—The syringe with asbestos packing should be used for making injections. Such an instrument is easily cleansed and boiled. I find the back just above the buttock the most convenient location in which to inject. The child can be easily held if this site is chosen. The parts should be carefully cleansed. The injection is given in the same manner as a hypodermic injection. The parts should not be rubbed after the injection.

EFFECT OF INJECTION.—There is a slight temporary rise of temperature following the injection. It is thought to be due to the entrance into the blood of the additional toxin contained in the antitoxin. This rise is succeeded by a gradual or critical fall, which continues until the temperature is subnormal. The membrane ceases to spread and exfoliates. In some cases these phenomena may be delayed twenty-four hours. The next day the pulse drops, the prostration gives way to a clear sensorium and good heart action, and sometimes the children sit up in bed and play with toys. The glandular swelling also diminishes markedly. In laryngeal cases if there has been threatened stenosis, the symptoms retrograde. Fully one-half retrograde spontaneously. On the other hand, if the temperature persists high after twenty-four hours and the membrane continues to spread, the injection should be repeated, especially if the swelling of the lymph-nodes is marked and there are symptoms of septic infection.

FIG. 56.



Antitoxin syringe with asbestos packing; can be taken apart and sterilized.

The effect of an injection of antitoxin on the blood is to diminish the number of leucocytes; just prior to the fall of temperature there is a critical hyperleucocytosis (Ewing, Schlessinger). Albuminuria continues, but this is also the case not only when no antitoxin has been used, but also in almost any infectious disease in which bacteria or their toxins circulate in the blood.

The eruptions which occur after the injection of antitoxin are of interest. At the site of the injection an abscess or phlegmon may form. This is the result of uncleanliness in technique or is due to some irritating substance in the antitoxin. A brawny erythema which gradually disappears may appear in a day or more at the site of injection. The injection may be rapidly followed by a painful eruption on the extremities, consisting of circumscribed violet colored spots, closely resembling erythema nodosum. The subcutaneous tissues are swollen, the joints are painful, and in addition there may be elevated temperature and a cardiac murmur. Herpes labialis and herpes nasalis, urticaria-like general eruptions, and morbilliform or scarlatiniform eruptions have followed injections. These eruptions appear from a few days to fourteen days after the injection.

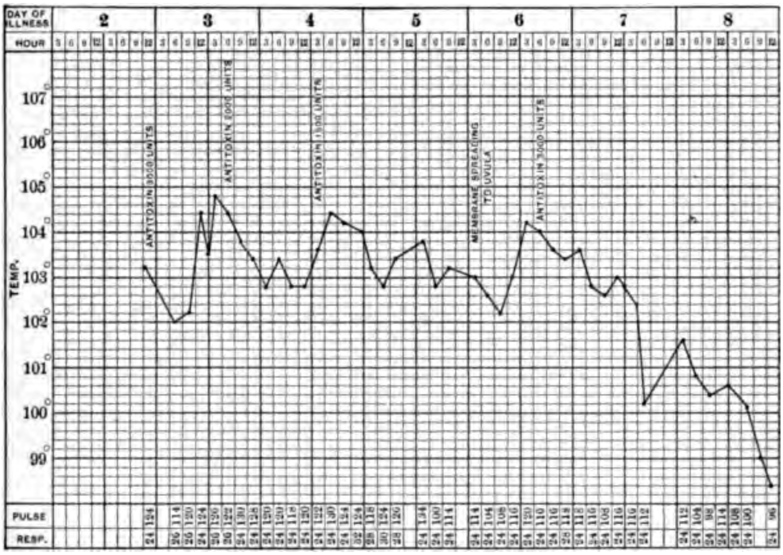
Conjunctival injection, tachycardia, and arrhythmia may be present.

The acute symptoms described above subside in most cases within two or three days.

Kidney irritation may follow the injection of large doses of antitoxin. In many of the cases reported, however, the renal symptoms have not been due to the antitoxin alone, and the same may be said of the recorded cases of endocarditis following antitoxin injections.

The introduction of antitoxin has by no means lessened the necessity of careful general management of a case by the physician. The temperature is controlled or modified by the ordinary hydrotherapeutic procedures. Antipyretics of the coal-tar series should not be administered, as they weaken the heart.

FIG. 57.



Septic form of tonsillar diphtheria: both tonsils and soft palate involved with nasopharynx. Persistence of temperature and recurrence of membrane after antitoxin injections on the sixth day. Injection of additional antitoxin, and critical drop of temperature thereafter. Recovery. Boy, six years of age.

If signs of cardiac paralysis of the early type set in, full doses of the cardiac remedies—digitalis (if the pulse is rapid), strychnine, caffein, camphor, and whiskey—are given. Of the remedies, digitalis must be used cautiously, else the pulse will be seriously depressed. Strychnia and caffein are the best and most available remedies. In the cases of cardiac irregularity it is best not to multiply drug remedies, or the stomach will be upset and the general conditions be aggravated. To a child three years of age we may give

$\frac{1}{150}$ grain of strychnia every three hours; whereas caffein is best used in the form of the citrate, a grain to a child of three years of age at similar intervals. The child is kept recumbent and the most assimilable forms of food are given, such as milk, kumyss, soft eggs, raw or boiled. In those cases in which there is gallop rhythm or extreme restlessness, digitalis in combination with morphin is given. To a child three to five years of age 2 minims of the tincture of digitalis may be given every three hours, and 1 or 2 minims of Magendie's solution by the mouth. The latter is repeated only when needed. In order to guard against cardiac weakness in the later period of the disease, a cardiac stimulant, such as strychnine, is given in small doses throughout the illness and in convalescence. The patient is not allowed to sit up too early should signs of cardiac irregularity appear at the outset of convalescence. In all cases of diphtheria the utmost caution should be exercised in reference to the heart.

The infant should not be nursed at the breast, lest the breast be infected. The milk should be pumped off and fed to the infant with a bottle. If there is diarrhœa, the milk is suspended and the bowel irrigated. The milk should not be resumed until all danger from this source is past. I administer alcohol in moderate doses if the prostration, pulse, and temperature warrant it. Infants under a year should be given half a drachm (2.0) of whiskey every three hours; infants more than two years of age, a drachm (4.0) at the same intervals. Diphtheria patients, especially those suffering from the septic form with constitutional symptoms, should be kept recumbent. The administration of remedies should not be forced, for struggling on the part of the patient may prove dangerous to the heart. During convalescence the whiskey may be replaced by wine. In these cases strychnine in small doses (grain $\frac{1}{200}$ [0.0003]) should be continued for some time, in order to support the heart. I advise a return to a mixed nutritious diet in all cases as soon as the temperature is normal; in this way the effect of the toxins on the tissues is counteracted as much as possible.

Some physicians still resort to the internal administration of corrosive sublimate in doses of grain $\frac{1}{100}$ (0.0006) or more, according to the age of the patient. It is given in the septic tonsillar and nasal cases, and also in the laryngeal forms of diphtheria.

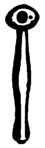
Local Treatment.—The presence of bacteria other than the diphtheria bacillus around the local lesions necessitates the use of local cleansing and disinfecting measures. In very young infants the nasal discharges are washed away by means of a glass syringe with a blunt rubber tip. The infant is laid on the side, and the nurse, standing behind the patient, irrigates the nostrils with normal salt solution at 110° F. (43.3° C.), as shown in Fig. 9. A pus basin is held underneath the chin. Older children will struggle, but by suasion they may be irrigated in the sitting posture. If there is much resistance, it is not desirable to insist on irrigation. In irrigating, the syringe should have a position parallel with the floor of the nasal fossæ. Spraying with a mild solution of Listerine or Dobell's solution is possible in some children, impracticable in others. The lymph-nodes, if slightly enlarged, are best treated by the application of warm oil of hyoscyamus; if very much swollen, the application of cloths wrung out in ice-cold water is of great utility. Small pieces of ice swallowed whole are grateful to the patient.

Treatment of Laryngeal Diphtheria.—In cases of mild laryngeal diphtheria an injection of antitoxin should be given. The patient should be placed under a tent, and grains x (0.6) of calomel sublimed every two or three hours, according to the necessities of the case. The efficacy of the calomel vapor is increased by passing steam into the tent at the same time. A convenient method is to place the calomel in a spoon, and heat the spoon over an ordinary candle, placed within the tent. The swelling of the larynx caused by the invasion of the Bacillus diphtheriæ and other bacteria is quickly relieved by the calomel, particularly in croupy cases with little or no membrane visible above the larynx. A tent may be improvised as described on page 65. Steam saturated with benzoin or thymol may also be passed into the tent. A croup kettle may be improvised from an ordinary teapot or one sold for the purpose may be employed. It is sometimes necessary to suspend the steam inhalations for an hour or longer, for the purposes of ventilation. The general treatment as to the heart, temperature, and food is the same as in the tonsillar forms of diphtheria. If signs of mechanical obstruction appear, intubation is indicated.

INTUBATION.—To Joseph O'Dwyer, of New York, belongs the credit of having perfected a method of relieving membranous obstruction of the larynx in diphtheria. Intubation in America and on the continent of Europe has completely displaced tracheotomy as a remedy for relieving laryngeal obstruction due to diphtheria.

Instruments.—Intubation tubes (Fig. 58) are of metal coated with rubber, though originally made of gilt metal. The tubes are graduated (Fig. 59) according to the age of the patient, and in their present form are the most ingeniously devised instruments ever given by American medicine to the world. The tubes are furnished with obturators, which fit into a handle, the introducer (Fig. 60). There is, in addition, a forceps (Fig. 61) with small departing blades, called the extractor. Finally, there is a gag (Fig. 62) so constructed that it may be introduced into the mouth and kept in position without obstructing the view of the operator.

FIG. 58.



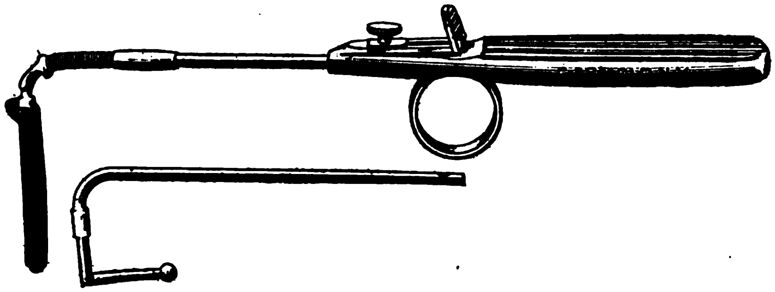
O'Dwyer tube.

FIG. 59.

Gauge for the age of the patient.

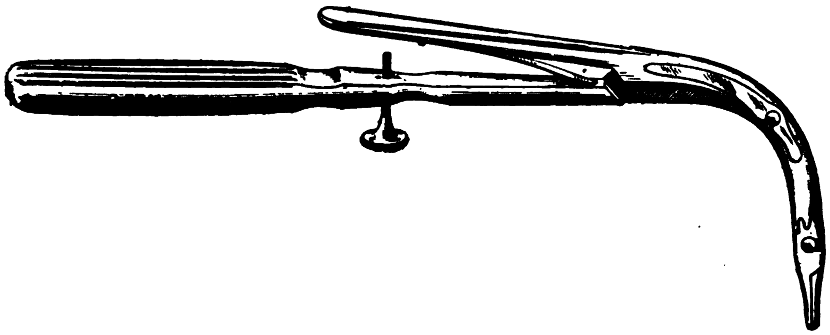
Indications.—We intubate when a progressive dyspnœa, which

FIG. 60.



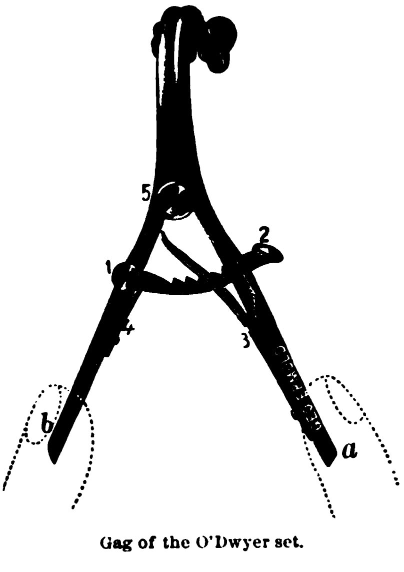
O'Dwyer tube, obturator, and handle.

FIG. 61.



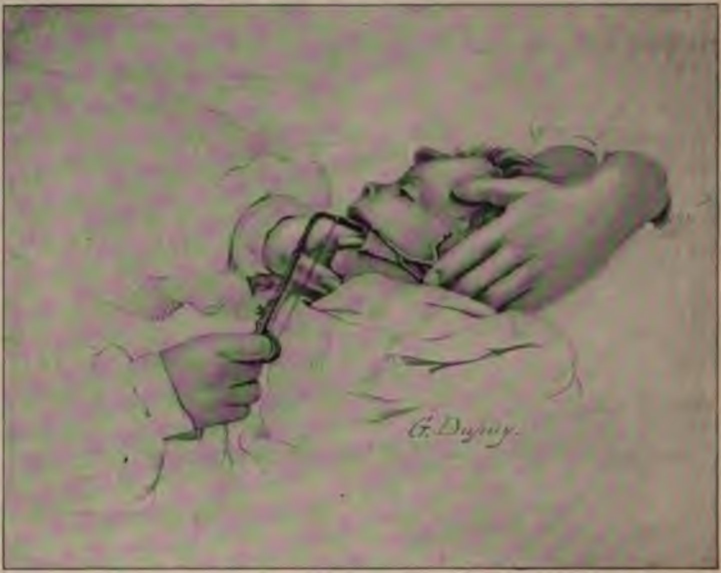
The O'Dwyer extractor.

FIG. 62.



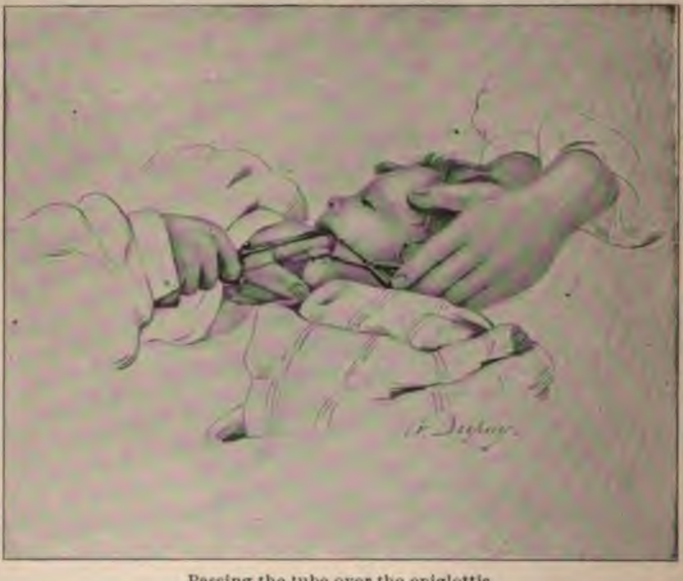
Gag of the O'Dwyer set.

FIG. 63.



Introduction of the tube along the index finger.

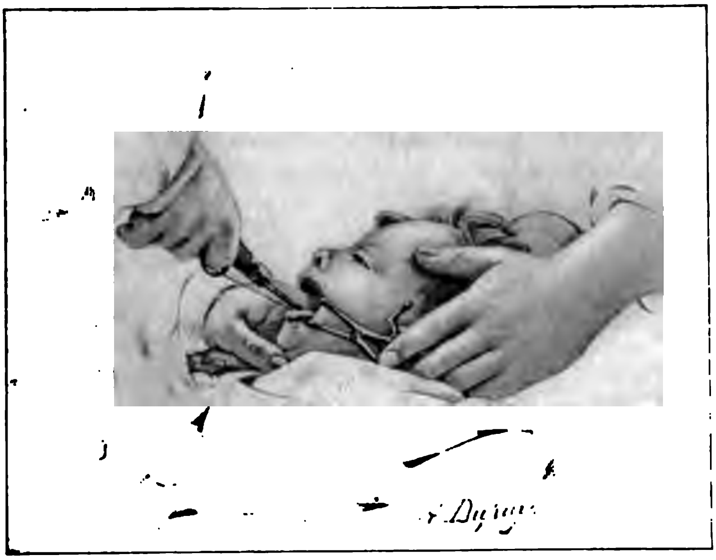
FIG. 64.



Passing the tube over the epiglottis.

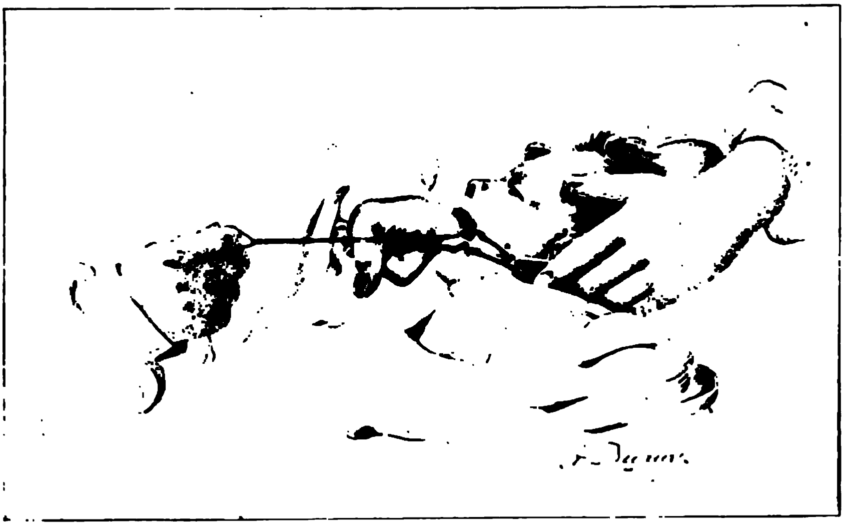
FIGS. 63, 64.—The operation of intubation of the larynx. Position of child, operator, an assistant.

Fig. 65.



Introduction of the tube into the chink of the glottis.

Fig. 66.



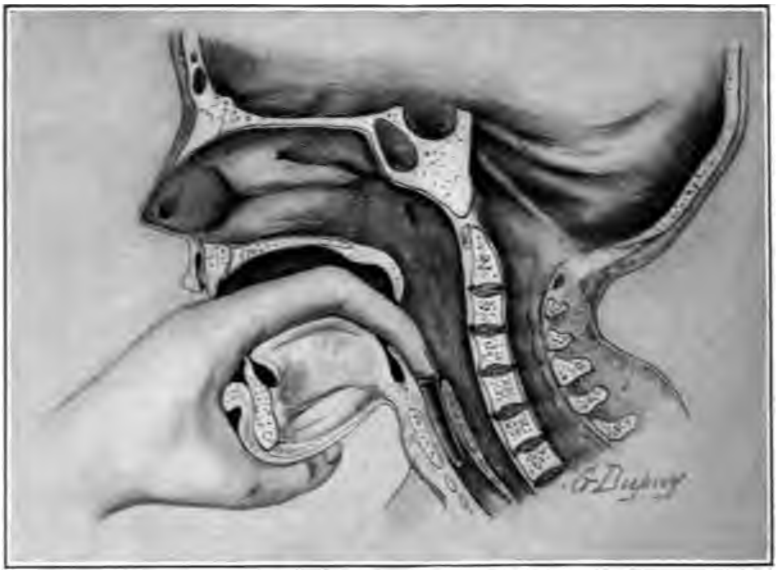
The index finger pushes the head of the tube into place in the larynx.

Figs. 65, 66.—The operation of intubation of the larynx. Position of child, operator, and assistant.

produces sensible exhaustion, exists. O'Dwyer never tubed the larynx except as a *dernier ressort*, and did not approve of early tubage. If an infant or child shows marked retraction of the suprasternal notch, retraction of the epigastrium, and stridor, with accompanying labored breathing, we should at once proceed to tube the larynx.

Mode of Operating.—The patient is wrapped in a blanket and held upright in the arms of a nurse, so that the head of the patient is on a level convenient to the operator, who stands facing the patient. An assistant standing behind the nurse steadies the head of the patient. The gag is introduced by depressing the tongue and jaw with a tongue-depressor. The assistant steadies the gag as he holds the head tilted very slightly backward. The tube, threaded with a silk ligature, is with its introducer held firmly with the right hand. The index finger of the left hand is now introduced into the mouth to the root of the tongue and search made for the epiglottis. In young infants[1] the epiglottis is short. The finger must be introduced quite deeply, feeling the arytenoid cartilages of the larynx, and is then drawn upward until the epiglottis is hooked forward. The index finger now holds the epiglottis (Fig. 67), and in a small larynx a skilled operator can also feel the arytenoids (Fig. 68). The tube is now introduced in the median line of the mouth along the

FIG. 67.



Method of hooking forward the epiglottis in intubation.

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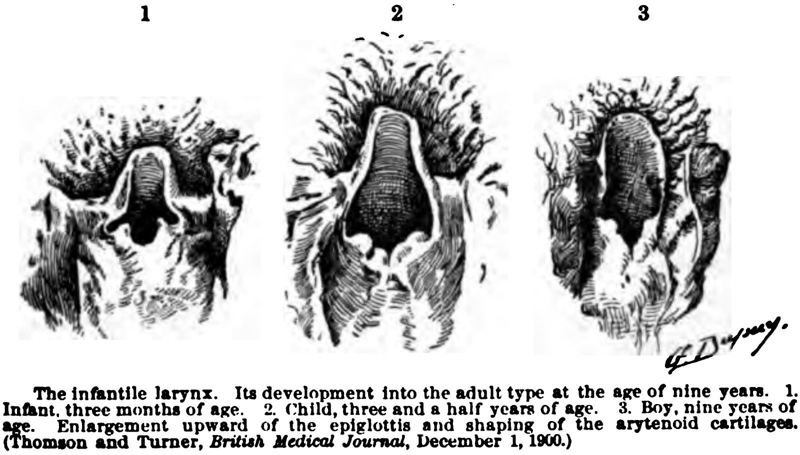
[1] *Peculiarities of the Larynx.*—Thomson and Turner have shown that the infantile form of larynx differs materially from that found later in life. At birth and in infants and young children the epiglottis is very small and gutter-shaped. The glottis is guarded above by the aryteno-epiglottic folds, which are closely approximated to each other. Toward the tenth year the epiglottis becomes much flattened, the aryteno-epiglottic folds become widely separated, and the larynx assumes the adult type. It is important to remember these points in the operation of intubation.

palmar surface of the index finger (Fig. 63), and the finger guides the tube over the epiglottis and into the chink of the glottis and prevents its slipping into the œsophagus (Fig. 64). The instrument should always be kept in the median line. The index finger holding the epiglottis should be held well to the angle of the mouth, so as to obtain plenty of room. No force should be used, else false passages will be made. If the first attempt at introduction does not succeed, we should not persist too long, but remove the introducer rapidly and give the larynx a few moments to recover its action, and then try again. As the tube passes into the chink of the glottis the handle of the introducer is elevated, as in Fig. 65, causing the end of the instrument to lie against the base of the tongue. The tube is released, the introducer and obturator withdrawn, and the index finger gently presses the head (Fig. 66) of the tube into the larynx. The gag is withdrawn, and the silken thread passed over the ear of the patient and fixed back of the ear with a piece of rubber plaster. Some operators remove the thread after ten minutes. The advantages of leaving the thread are that, should the tube be coughed up in the absence of the physician, it can be recovered by the nurse. In extubating, it is an aid in removing the tube.

No anæsthetic is required, and ordinary assistance only is necessary. The air passing into the bronchi is moistened in its passage through the natural passages. The danger that food particles may pass into the larynx has been exaggerated. The detachment of membrane in front of the tube is very infrequent. Should it happen, and the membrane not be expelled on removal of the tube, tracheotomy is admissible if asphyxia is imminent. It sometimes happens that the tube is expelled many times after introduction. It should be reintroduced or a larger tube employed.

FIG. 68.



The infantile larynx. Its development into the adult type at the age of nine years. 1. Infant, three months of age. 2. Child, three and a half years of age. 3. Boy, nine years of age. Enlargement upward of the epiglottis and shaping of the arytenoid cartilages. (Thomson and Turner, *British Medical Journal*, December 1, 1900.)

If the operator has chosen to leave the silken cord of the tube *in situ*, it should be passed through the space between the first molar and bicuspid tooth, to avoid its being gradually bitten through. Should it be bitten through, the finger is introduced into the mouth to the top of the tube and the thread withdrawn, while the tube is kept in the larynx with the finger.

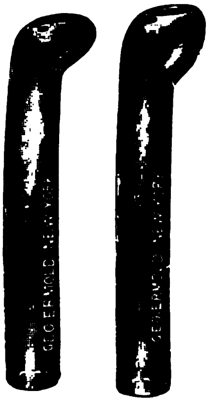
The tube is allowed to remain from twenty-four hours to five days. Since the introduction of antitoxin the tube is taken out much sooner than was formerly the practice. If there is marked improvement in two or three days, removal of the tube should be attempted and the effect of such a procedure on the breathing should be observed.

Extubation.—The patient is placed in the same position as for intubation. The left index finger is passed into the mouth and search made for the epiglottis, the tip of the finger resting on the arytenoids. The extractor is passed along the palmar side of the finger and is guided into the opening in the tube by the tip of the finger. Extubation is more difficult than intubation. The extractor should be regulated by means of a small screw, so that the blades do not open too far. This is to guard against injury to the soft parts of the larynx should the opening of the tube not be entered.

Dangers.—The dangers of intubation include detachment of membrane during introduction, laceration of the parts, the formation of false passages, and asphyxia. The first rarely occurs unless force is used. The second can only occur as a result of rough and unskilled efforts at intubation. The third occurs only following prolonged efforts at introduction of the tube. Even a skilful operator may pass the tube into the ventricle of the larynx. Northrup has published a case in which there was a false pocket above the cords which prevented the entrance of the tube into the larynx. In other cases there is what is described by O'Dwyer as subglottic stenosis. Northrup thinks that this is due to swelling of the mucous membrane at the level of the cricoid cartilage. In these cases introduction of the tube is very difficult. The operator may be compelled to use force to push the tube past the stenosis or a smaller tube may be employed. While the tube is being worn, it may become obstructed by membrane. This is denoted by a return of the croupy cough, a snarling, flapping sound, and obstruction to expiration. To obviate these difficulties, O'Dwyer has had short tubes constructed without a retaining flange. These tubes have a special introducer. The largest size for the age is chosen, and the tube forced into the larynx. These tubes should be used only by skilled operators. The tubes are allowed to remain but a short time in the larynx. Other complications are the formation of granulations or ulcerations around the lower end of the tube if it is too long, and at the cricoid cartilage if it is too large. The former condition is not serious; the latter may destroy the cartilage. Granulations may form about the head of the tube. In this case tubes with built-up heads are used to press on the granulations, thus causing them to atrophy (Fig. 69).

Feeding the patient after introduction of the tube requires care. Most infants will nurse with the tube in the larynx. In some there is considerable difficulty in swallowing. The patient is taken in the lap of the nurse and fed with the head held a little lower than the body. Fluids thus cannot enter the trachea and cause pneumonia.

FIG. 69.



Built-up tubes.

Treatment of the Complications.—The treatment of the BRONCHO-PNEUMONIA which complicates diphtheria is similar to that employed in the treatment of the primary affection. The question of the further administration of antitoxin always arises in these cases. I give it in full doses, since it is known that the Bacillus diphtheriæ is the causative factor.

The GASTRO-ENTERITIS which complicates diphtheria is apt to prove a very serious complication. It should receive the same treatment as a primary gastro-enteritis.

Both the severe and the mild cases of diphtheria of the vulva or of the vulva and vagina should be treated with antitoxin. In some of the mild forms of undoubted bacillary origin which I have seen, the membrane was easily removable. In these cases, contrary to the practice in the tonsillar cases, I remove the membrane with a spud wrapped with cotton. The bleeding surface left after removal is painted with a 10 per cent. solution of silver nitrate once daily. I have cured cases by this method alone. If there are extensive swelling, necrosis, and gangrene, this method will be of no avail, and antitoxin should be given in full doses, and repeated according to indications.

PARALYSES.—The treatment of diphtheritic and especially postdiphtheritic paralyses is at present largely empirical. The symptoms appear with the degenerations in full progress. Of all the remedies recommended, Fowler's solution in tonic doses has seemed to give the best results. I have seen patients recover when given arsenic, nutritious food, and abundant fresh air. Hypodermic injections of strychnine are of questionable value. Electricity is of value as an adjuvant to massage of the muscles only in general paralysis. It is questionable whether in some cases it is not capable of doing great harm by tiring nerve and muscle. I find that patients do very well with hydrotherapy and massage. In these cases the last reaction to reappear is the patellar reflex.

Diphtheroid.

(*Pseudodiphtheria; False Diphtheria.*)

The term diphtheroid includes all pseudomembranous formations not caused by the Klebs-Löffler bacillus. It was first proposed in 1860 by Boussage, and has recently been adopted by Weigert, Escherich, Heubner, and Behring.

Occurrence.—This form of pseudomembranous formation is most frequently met with in the exanthemata, especially scarlet fever and measles. In the former it is a common complication. It is also met in other conditions, and fevers such as typhoid, and may occur as a primary affection.

Etiology.—The cases met in the exanthemata were first described by Prudden, who believed that the process was due to a streptococcus, the Streptococcus diphtheriæ. Since then, the occurrence of the streptococci has been confirmed, but there have also been added to this group of pseudomembranous inflammations cases in which the pseudomembrane is caused by a diplococcus, the so-called Roux coccus. The pneumococcus (Jaccoud and Menetrier) may also cause a pseudomembranous angina. The Bacterium coli and the gonococcus (the latter in newly born infants) may cause a membranous formation in the mouth and throat. The Staphylococcus pyogenes aureus is also found in these diphtheroid membranes. By far the most important group is that first mentioned, the pseudomembranous or diphtheroid inflammation caused by the Streptococcus pyogenes, which is none other than that isolated by Prudden. These cases are characterized by their favorable course; while the mortality in diphtheria varies from 20 to 35 per cent., according to the age of the patient, the virulence of the epidemic, and the early administration of antitoxin, the mortality of the diphtheroid cases ranges from 3 to 5 per cent. (Park, Baginsky).

Symptoms and Course.—The pseudomembrane occurs on the tonsils, pharynx, and larynx. There are adenopathy and fever. The prostration and constitutional disturbance are much less than in true diphtheria. Membranes and casts of the larynx and trachea may be expelled. Suppuration of the lymph-nodes may also occur. In many of these cases there is a complicating bronchopneumonia of the streptococcus type (Prudden and Northrup), which usually results fatally.

Diagnosis.—It is not possible to make a diagnosis of diphtheroid from the gross appearance of the membrane. The culture-test is the only reliable method of determining the nature of a pseudomembranous exudate. If the first culture gives a negative result, a second one should be made.

Treatment.—Clinically the treatment is much the same as in true diphtheria. The administration of antitoxin should not be delayed until the nature of the exudate is determined. It is then discontinued. An exception to this rule may be made in the scarlatinal form of diphtheroid, in which it is safe to wait for the result of the culture-test, unless it is known that the patient has been exposed to diphtheritic infection. In such a case antitoxin should be administered. In laryngeal obstruction the indications for treatment are the same as in true diphtheria.

XVI. SCROFULA or SCROFULOSIS.

Scrofulosis is a constitutional dyscrasia which occurs chiefly in childhood, and is characterized by enlargement of the lymph-nodes and slow, sluggish inflammation of the mucous membranes, skin, joints, and bones. There is a distinct tendency to repeated inflammations from very trivial causes.

Occurrence.—Scrofulosis is almost exclusively a disease of childhood and youth, and is rarely seen after the twentieth year. Henoch and Birch-Hirschfeld state that the majority of cases occur between the third and the fifteenth year. Females are more frequently affected than males. Ruhl found it to be most common between the sixth and the tenth year.

Forms.—Cornet and Ponfick recognize three forms of scrofulosis:

a. The tuberculous form, which is practically identical with cutaneous, lymphatic, and bone tuberculosis.

b. The pyogenic form, in which the tubercle bacillus is absent in the lesions or products of inflammation, but which in its outward clinical manifestations closely resembles the tuberculous form. In this form the essential etiological factors are the Staphylococcus pyogenes aureus and albus and the Streptococcus pyogenes.

c. The mixed form, in which both the tubercle bacillus and the pyogenic bacteria are found in the lesions and products of inflammation.

Etiology.—In considering the etiology of scrofulosis, it should be borne in mind that at the period of life during which the disease occurs the lymph-nodes are not structurally fully developed. On account of this condition and of deficiencies of other tissues such as the skin and mucous membranes, bacteria obtain easy access through the skin, mucous membranes, and lymph-vessels even when there is no breach of continuity of surface (Cornet).

It is also true that certain individuals, once infected, show an inherited predisposition to affections of the mucous membranes and other tissues. This does not mean that scrofulosis as such is hereditary, but that in these subjects exposure to the essential causes of the disease will result in permanently establishing the conditions.

The essential causes of scrofulosis are the tubercle bacillus and the pyogenic bacteria just mentioned. These bacteria are present in ill-ventilated rooms occupied by phthisical patients. Scrofulous infection may be traced to parents, brothers, sisters, nurses, and playmates. Dried sputum is a prolific source of infection. Infection is favored by any solution of continuity of the skin or mucous membranes, and also by hyperæmia or œdema of these tissues.

The predisposing factors are social conditions, unhygienic surroundings, moist dark dwellings, uncleanliness, improper or insufficient food, and lack of fresh air and exercise. The overcrowding in the poorer quarters of cities affords abundant opportunities for infection. Any weakening of the system by infectious diseases, such as measles, pertussis, scarlet fever, diphtheria, rachitis, struma, cretinism, and erysipelas, may be the starting-point for infection. Traumatism or frostbite favors the entrance of bacteria.

Morbid Anatomy.—The gross pathological changes are the same in both the pyogenic and tuberculous forms of scrofulosis.

The Pyogenic Form.—The mucous membranes are the seat of hyperæmia and thickening. There are increased secretion and activity of the glands, also desquamation of epithelium, and excretion of serum and blood-elements from the surface of the membrane. Adenoids, enlarged tonsils, bronchitis, intestinal and vaginal catarrh, are the most common of the lesions of the mucous membrane.

SKIN.—There are eczema, thickening of the epidermis, and transudation of serum and elements of the blood (erythrocytes and leucocytes).

The CORNEA shows conjunctivitis and phlyctenulæ.

The LYMPH-NODES show hyperplasia, which is scarcely noticeable in the early stages. They subsequently enlarge to form tumor masses, which may soften as a result of suppuration or may retrograde to the normal.

The Tuberculous Form.—SKIN.—Lupus is the form of change found in the skin.

LYMPH-NODES.—The nodes in almost any part of the body may be involved. They are enlarged to a greater or less degree, and are infiltrated with tubercle. On section they show either simple caseation or mixed infection. The latter is the case if pyogenic infection is combined with the tuberculous form. Nodes which are the seat of cheesy degeneration may soften and break down, forming cold abscesses. These may open externally or into the bronchi, bloodvessels, pericardium, or peritoneum.

JOINTS AND BONES.—In the bones the tuberculous invasion gives rise to fungous or dry caries. Several such foci may be present in the same bone. These foci may heal and years afterward become inflamed as a result of traumatism or infectious disease.

The diaphyses and extremities of the long bones are thickened as the result of periosteal inflammation. The ends of the bones are the seat of tuberculous [illegible]. The joints may be involved. At first there is serous exudate without [illegible] into the joint of the tuberculous foci. Later there are thickening of the synovial membranes and seropurulent exudate into the joint-cavity, with destruction of the cartilages and heads of the bones.

Symptoms.—General Clinical Picture.—The patient is anæmic, but not necessarily emaciated; on the contrary, there is a very good panniculus of fat in the majority of cases. The face of some of these subjects presents an eczematous or lupoid eruption. The lips are thick; the conjunctivæ may be injected, and there may be blepharitis or phlyctenula of the cornea. Snuffles and nasul catarrh or ozæna are present. The majority of the patients are mouth-breathers, and suffer from adenoids and enlarged tonsils. In some there is chronic otitis with an offensive discharge. There is a fulness about the neck due to enlarged lymph-nodes. The body may present skin eruptions in the form of ecthyma or varieties of eczema. The general surface is in other cases free from eruption, is pale, and has a transparent, marble-like appearance, showing the blue veins underneath. Many of these patients give a history of chronic bronchitis. In others the remains of old suppurations of the lymph-nodes about the neck are seen in the form of livid cicatrices. If the long bones of the extremities have been affected, the surface of the skin shows either old or recent bone sinuses. The symptoms in most cases develop first on the skin and mucous membranes; the lymph-nodes then enlarge, the bones and joints are next involved, and finally, if the case does not progress favorably, amyloid degeneration of the different organs and emaciation develop as a result of prolonged suppuration. In all cases the changes in the lymph-nodes play a leading part, and are characteristic.

THE SKIN.—In the tuberculous variety lupus is the most common form of skin lesion; in another form there is the so-called scrofuloderma of Besnier. Lichen scrofulosorum, with the characteristic enlargement of the lymph-nodes, is another form of skin eruption. In the pyogenic variety eczematous and acneform eruptions are present. In such cases the skin is thickened as a result of chronic inflammations. There are suppurating rhagades around the eyes, mouth, and anus, and ecthymatous eruptions may be present on the trunk and extremities. A form of scrofulous ecthyma, made up of purple painful nodules resembling erythema nodosum, has been described by Hutchinson. Hebra has described a prurigo of the scrofulous subject.

MUCOUS MEMBRANES.—There are ulcerations and chronic catarrh of the nasal and bronchial mucous membranes, and in some cases ozæna of an atrophic character. These patients have adenoids and enlarged tonsils. The tonsils are favorite seats of infection. In other cases the posterior nasal and pharyngeal catarrh leads to retropharyngeal abscess, or caries of the spine may cause abscess formations in the retropharynx.

THE EARS.—As a result of the catarrh of the nasopharynx chronic otitis may develop. When otitis follows any of the exanthemata in a patient with scrofulous tendencies, it pursues a chronic painless course. Such an otitis may tend to tuberculous disease of the mastoid with sinus thrombosis, or even to tuberculous meningitis. There is pain only when there is a mixed pyogenic infection.

THE EYE.—Chronic eczema of the lids, blepharitis, phlyctenula of the cornea, and keratitis fasciculosa are seen. The phlyctenulæ do not yield readily to treatment. Hypopyon of the anterior chamber may also be present. Trachoma is in some instances of a tuberculous origin. Lupus of the conjunctiva is sometimes present.

LYMPH-NODES.—The tuberculous and pyogenic forms of enlargement of the lymph-nodes are at the outset similar. The pyogenic varieties are associated with enlarged tonsils and adenoids. The skin over the enlarged nodes may remain normal for months or years, or in both the tuberculous and pyogenic varieties it may become adherent, red, inflamed, and break down. The lymph-nodes discharge, leaving suppurating cicatricial openings.

Clinically, infections of the scalp lead to enlargement of the lymph-nodes of the neck and retromaxillary region. Those of the cornea, iris, and ear tend to enlarged preauricular nodes and to enlarged nodes of the submaxillary region. Infections of the mouth and tonsil cause enlarged nodes at the angle of the jaw and beneath it. Otitis with mastoid disease causes enlargement of the node on the point of the mastoid. The lymphatics of the gums and lips are connected with the nodes of the submaxillary region and angle of the jaw. Affections of the nose will cause enlargement of the glands of the neck (Jacobi). Lesions of the fingers will result in enlargement of the cubital and axillary nodes. Infection of a circumcision wound or balanitis will cause enlargement of the inguinal lymph-nodes, as will also infections of the foot and knee.

The lymph-nodes in direct line are always involved; distant ones are never infected unless there is infection of the intermediate nodes. It was formerly believed that the bronchial nodes were particularly subject to infection. Any special susceptibility to infection shown by these nodes is due to their location, infectious material being frequently present in their vicinity.

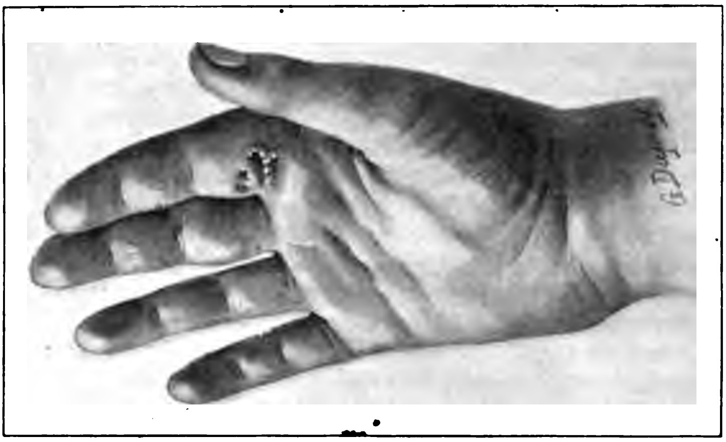
Cornet found the bronchial nodes affected in 103 out of 126 cases of tuberculous disease occurring before the completion of the fifteenth year. These observations confirm the statement of Henoch, that the bronchial nodes are affected in the majority of cases of tuberculous disease. There are no statistics showing the involvement of lymph-nodes in the pyogenic forms of scrofulosis. Becker, Barthez and Rilliet, Henoch, and Northrup have described the enlargement of bronchial nodes. According to Henoch, they may, even if tuberculous, be enlarged without involving the lung tissue.

By pressing on the vagi they may cause rapidity of pulse, and if on the recurrent laryngeal may give rise to spasmodic dyspnœa or to a croupy cough. Pressure on the œsophagus may cause dysphagia; pressure on the trachea may cause inspiratory dyspnœa; and pressure on the pulmonary veins, hyperæmia of the lungs. Henoch and Baginsky doubt the possibility of diagnosing these enlarged nodes even with the help of all these symptoms.

These nodes may retrograde to the normal size (West) or they may break down and perforate into a bronchus or the trachea. If they perforate into the pericardium, pleura, or mediastinum, inflammation results at these points.

The mesenteric lymph-nodes may enlarge and cause pain or tuberculous infection of the peritoneum (tabes meseraica). In some cases they may be palpated through the abdominal wall.

FIG. 70.



Tuberculosis of the proximal phalanx of the index finger in a scrofulous child the subject of extensive lupus of the face and extremities ("Spina ventosa").

THE BONES AND JOINTS.—The extremities of the long bones are most frequently the seat of disease; the diaphysis rarely so. The phalanges of the fingers, the toes, the radius, the ulna, and fibula, are affected in the order of naming. The joint-cavities may at first contain exudate without perforation of the cartilage; later, pus is found in the cavity.

All of the structures of the joint are involved, and the joint may eventually be destroyed. Suppuration of a chronic nature may, as stated elsewhere, tend to amyloid degeneration of the liver and spleen.

There is, in addition, a progressive anæmia. The temperature is sometimes raised a half or three-quarters of a degree above the normal, at others it is normal. Exhausting sweats occur; the disturbances of nutrition become in some cases extreme. There may be intestinal diarrhœa.

Course and Prognosis.—This condition is not necessarily fatal. Many cases make a good recovery under proper management. This is particularly true of the pyogenic form. The tuberculous variety may also retrograde if the disease be localized to certain lymph-nodes or bone foci.

Diagnosis.—The diagnosis of the pyogenic form is made from the clinical history; that of the tuberculous variety, either from the presence of the tubercle bacillus in the pus or lesions of the disease. There are mixed forms in which it is not always possible to decide whether the process is tuberculous or pyogenic. The clinical history and blood examination will be of service in differentiating scrofulosis from leukæmia, pseudoleukæmia, and lymphomata of a malignant nature (Plate XIII.). In other cases the diagnosis from late forms of hereditary syphilis cannot always be readily made. The history of such cases is of importance. In many cases a resort to antisyphilitic treatment will be necessary to complete the diagnosis.

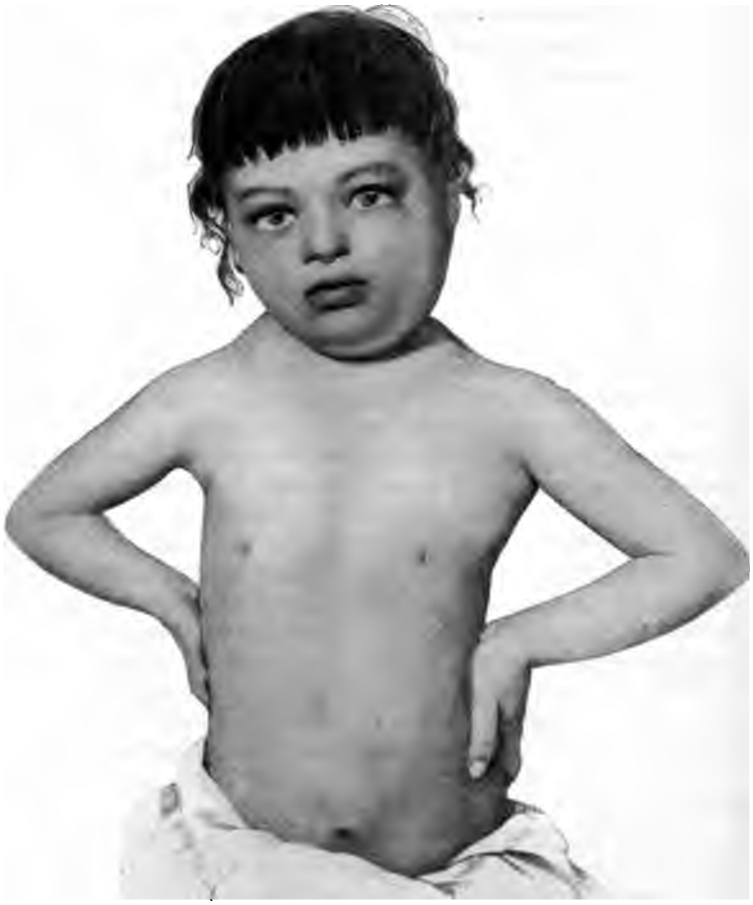
The **treatment** of scrofulosis is directed toward limiting if possible the spread of the infection, preventing reinfection of the patient, and instituting local treatment of the lesion. In order that the disease may be treated successfully, the patient should be placed under hygienic surroundings. If the patient is in the city, removal to the country is advisable. The food should be plain and nutritious; milk, eggs, meat, vegetables, and cereals should form the diet. The hygiene of the skin is important. Alkaline or sea baths give tone to the skin. Moderate exercise in the open air is also of great service in correcting the anæmia and tendency to inaction shown by these patients. In a word, the patient should be removed from the conditions and surroundings which originally induced the infection.

The medical treatment is limited to the exhibition of such tonics as iron, Fowler's solution, and strychnine. The intestines should receive attention during the administration of iron. Fowler's solution gives better results in pyogenic lymphadenitis than in the tuberculous form. The syrup of ferric iodide in full doses has a tonic effect on the mucous membranes. Baginsky advises the exhibition of preparations of thyroid gland. I have not seen any markedly good results obtained by this method of treatment.

Cod-liver oil is of great value in this disease. In the form of emulsions it should be given in full doses; with young children its use must sometimes be suspended on account of the laxative effect on the intestines.

The local skin lesions should receive appropriate treatment, as should also the bones, joints, and suppurating lymph-nodes. It is not

PLATE XIII.



Multiple Lymphosarcomata as Differentiated from Scrofulous Lymphadenitis. Enlargement of the cervical, supraclavicular, and axillary lymph-nodes. Child six years of age.

within the province of this work to enter upon the surgical details of such treatment.

XVII. TUBERCULOSIS.

Etiology.—The essential cause of tuberculosis is the tubercle bacillus. This micro-organism gains entrance to the body through the respiratory channels (inhalations), the intestinal canal, and through wounds. The air in ill-ventilated rooms occupied by tuberculous individuals may at times contain the bacillus in particles of dried sputum. Any abrasion of the mucous membrane of the respiratory tract affords opportunities for the entrance of the bacillus into the blood and lymph-channels. Among cases of this kind belong those in which phthisical individuals have breathed into the mouths of asphyxiated infants or children.

Not much importance is attached by certain authors to the possibility of infection through the intestinal canal (Baumgarten, Bollinger). However, cases have been reported in which infected cows' milk has caused tuberculosis in infants (Leonhardt, Sonntag, Eber). Infection in this manner is rare, because the food of infants (milk) is as a rule heated before it is taken into the stomach.

In a recent monograph Blackader says that of 125 autopsies on tuberculous children Northrup found that in 88 cases the primary infection had occurred through the respiratory tract. In 3 cases the pathway of infection was the intestinal canal, and in 35 cases the primary seat of infection remained undetermined. Of 75 additional autopsies, Bovaird found the primary lesion in the lungs or bronchial nodes in 60 cases. English writers, according to Blackader, place the frequency of primary infection through the intestinal tract at a much higher figure (25 to 30 per cent.) than do American investigators.

Still, of Great Ormond Street, gives the following statistics of primary tuberculosis in infants and children :

Lung	105	138	153=57 per cent.
Probably lung	33		
Ear	9	15	
Probably ear	6		
Intestine	53	63=23.4 per cent.	
Probably intestine	10		
Bones, etc	5	53=nearly 20 per cent.	
Fauces	2		
Uncertain	46		

German pathologists maintain that primary tuberculosis of the intestinal canal is rare. Marfan, the French pediatrist, places the frequency of primary intestinal tuberculosis at 87 per cent.

In general, it may be said that the avenue of infection in infants and children is through the respiratory tract. Infection through infected milk is now believed to be very rare. The importance of this mode of infection has been heretofore much exaggerated (Blackader).

Infection through mother's milk has been recorded as occurring in the newborn infant (Roger and Garnier). Heredity only creates predisposition to the disease, as it does with adults. Dennig found that 58 per cent. of his cases of tuberculosis occurred in children of tuberculous families.

Infection through wounds is very uncommon with children, because they are not usually exposed to traumatism. It is not so likely to result in general tuberculosis as infection through the other channels. This is proved by the fact that in the adult so-called autopsy tubercle rarely tends to general infection. Infants who are subjected to ritual circumcision are exposed to infection through the practice of arresting the resulting hemorrhage by mouth suction. I have seen eight instances of such infection. In these cases there is a primary enlargement of the inguinal lymph-nodes before the process becomes general. The vertebræ may become tuberculous or meningitis of a tuberculous nature may result.

The infectious diseases play an important rôle as predisposing factors in tuberculosis. Measles, scarlet fever, pertussis, and influenza, by lessening the resistance of the economy and impairing the integrity of the air-passages, favor the infection. Tuberculous bronchopneumonia occurs under these conditions, either because the tubercle bacillus was present in the body before the infection was contracted or gained access subsequently (Fränkel). In the majority of cases the former condition is the rule. Cold, unhygienic surroundings, and poor food, all predispose to infection as with adults.

Fœtal Tuberculosis.

Fœtal tuberculosis is rare, but undoubted cases are recorded in the literature. Jacobi's case was that of a seven months' fœtus born of a tuberculous mother, which showed miliary tuberculosis. Including this, there are 6 cases of undoubted fœtal tuberculosis recorded in the literature (Jacobi, Birch-Hirschfeld, Lehman, Schmorl, Kockel, and Wollstein). In all these cases the mode of infection was by way of the placenta.

Of the cases occurring in very early infancy and the newborn, very few exist in the literature which may be traced to intrauterine infection, and are therefore to be considered as congenital. In these cases the children died so soon after birth, and the lesions were so far advanced, as to justify this assumption. Tubercle bacilli are exceedingly rare in the testis or sperma, and it is questionable whether in such cases a tuberculous fœtus can result. In the human subject there is not one authentic example of infection through the sperma of a tuberculous individual. Among animals we find that there are many cases of observed intrauterine infection; but no cases in the human subject of infection brought about by insemination of the male. There are 70 cases in animals, which have been carefully sifted, of fœtal tuberculosis of the congenital variety. In all the avenue of infection was by way of the placenta. In no case was the ovum primarily infected or the germinative infection traced to the father. True congenital tuberculosis, therefore, in the sense just intimated, is rare. Fœtal tuberculosis occurs, as shown above, but is not such an important mode of infection for so widespread a disease as tuberculosis.

There is another form of fœtal tuberculosis, and that is the so-called bacillosis or bacillary form. In this form the fœtus is found to be free from the lesions of tuberculosis, but bacilli are found in the umbilical vein or in the liver or in the fœtal organs. Such are the cases, including that of Bugge, of fœtal tuberculosis without lesions. The rarity of the tuberculosis of the fœtus is due to the fact that bacillosis of the mother is rare. Bacilli occurring free in the circulation in advanced phthisis is rare in itself; and they soon become localized in the tissues. The placenta, as also the liver of the fœtus, acts as a barrier and filter of the tubercle bacilli, or they die in the blood-stream. Another reason, as stated, is the rarity of congenital lesions of the mother whereby the ovum may be infected.

The characteristics of fœtal tubercle are: (1) That it is rarely pulmonary. The liver is frequently affected, also the spleen, kidneys, and suprarenal capsules; whereas in the lungs only isolated tubercles are found. (2) Fœtal tissues are a favorable soil for tubercle. (3) Giant cells are wanting. (4) Bacilli may be present in large numbers without the development of gross lesions (bacillosis without lesions).

Under placental infection are to be included those cases in which the tubercle bacillus has been found in the blood of the fœtus without accompanying changes in the organs (Schmorl), and those in which tubercle nodules and enlarged lymph-nodes have been found at birth (Landouzy and Lehman). In both these forms of tuberculous infection the mother had suffered from acute miliary tuberculosis. The spermatozoön and testis may contain tubercle bacilli in the absence of gross tuberculous lesions of the organ (Nakarai and Kockel). Tuberculosis may in this way be conveyed into the uterus at the time of conception. Jahni and Weigert found tubercle bacilli also in the Fallopian tubes of women dying of phthisis, although there were no gross changes in the tubes. The ovum may thus convey tubercle bacilli. As a rule the bacilli thus introduced into the ovum of the fœtus are dormant during intra-uterine life. They may develop at any period after birth (Baumgarten). Additional facts supporting the theory of intra-uterine infection of the fœtus have been reported by Baumgarten and Roloff, who found a cheesy nodule in a dead-born fœtus, and by Birch-Hirschfeld and Bugge, who found bacilli in the blood of a fœtus born of a tuberculous parent.

The **frequency** of the occurrence of tuberculosis in infancy and childhood varies with the environment. Statistics are therefore only relative. Seidl found that of 646 consecutive autopsies upon children, 14 per cent. were tuberculous. Forty-five per cent. of all the cases of tuberculosis occur during the first two years of life, 25 per cent. of the total number occur during the first year, and 71 per cent. during the first five years (Queyrat, Lannelongue, Dennig). It is slightly more frequent among girls than boys.

Pulmonary Tuberculosis.

Seventy per cent. of the infants and children who die from tuberculosis show lung-changes (Dennig). Infection first occurs through the respiratory tract. A cheesy lymph-node may burst into the bronchi, and bacilli may thus gain access to the lung alveoli and cause changes, as they do in the adult lung. Hæmatogenous infection occurs through the bursting of a small tuberculous nodule into a bloodvessel, thus flooding the lung with infectious matter, or by the carrying of minute emboli of this material to distant parts of the lung.

Tuberculous bronchial lymph-nodes, bone, and pleura may also give rise to infection of the lung through the lymph-channels. The part played by the infectious diseases in its dissemination has been already mentioned.

Morbid Anatomy.—The three principal forms of tuberculosis of the lungs which occur in infants and children are:

The **miliary form,** which is characterized by the eruption of miliary tubercles throughout the lung. The lung is on section found to be dark red, hyperæmic, and to contain less air than the normal lung. The bronchial mucous membrane is hyperæmic and covered with blood and mucus.

The **cheesy or cheesy ulcerative form,** also called florid phthisis, takes the form of cheesy lobar or lobular pneumonia. In recent cases the lung is grayish red, and there are areas which rapidly become cheesy, and are not encapsulated. These may coalesce, involving the greater part of a lobe in the process. Small cavities are frequent, large ones rare. The cheesy ulcerative form occurs as a result of the aspiration of large numbers of tubercle bacilli.

The **chronic form,** which is a cheesy fibrous bronchopneumonia, is essentially a tuberculous bronchopneumonia. Round cheesy nodules are found surrounded by a fibrocellular zone resulting from the destruction of extensive areas of lung-tissue. The pulmonary pleura is thickened. The bloodvessels participate in the process. There is endarteritis with miliary tubercle in the walls of the bloodvessels, and there may be thrombosis. The tubercles may burst into the interior of the bloodvessels. The bronchi, trachea, and larynx may be affected. There are ulcerations of the mucous membrane and destruction of cartilage. The bronchial lymph-nodes or glands are enlarged and infected in most cases of tuberculosis of the lungs in children. Henoch has, however, shown that the bronchial nodes may be tuberculous and greatly enlarged without involvement of the lung-tissues. Northrup found the bronchial lymph-nodes affected in 125 consecutive autopsies. The whole node is converted into a cheesy mass, which may soften and break down. If there is a perforation into a bronchus, masses of bacilli may be discharged into the lung. Perforation into the bloodvessels may also occur. The nodes may form small masses or large mediastinal tumors at the root of the lung.

Localization.—The apices of the lungs of infants and children are not as in adults the region most frequently affected by tuberculosis. The first change may appear in the lower lobe or the lower portion of the upper lobe, and spread thence. This is accounted for by the miliary character of the affection in the lungs of infants and children (Rindfleisch), and also by the fact that in many cases the process spreads from the bronchial nodes or glands to adjacent parts (Weigert).

The **symptoms** of tuberculosis of the lungs in infants and young children are not so characteristic as in the adult, nor is there a gradual development of the symptoms pointing to involvement of the lungs. After the fifth year of life the symptoms closely resemble those seen in the adult. As regards infants, we shall describe only clinical types of the disease. Even these exhibit many varieties.

Henoch has described forms of tuberculosis in infants which closely resemble cases of marasmus due to gastro-enteric disease. In many of them there are steady emaciation and progressive muscular weakness; the infant lies helpless; the abdomen is retracted; the eyes may present a conjunctivitis; the cervical, axillary, and inguinal glands may be slightly enlarged; there is constipation alternating with diarrhœa; the skin is easily inflamed and abscesses may form. In the terminal period vomiting sets in. The lungs throughout the course of the disease may present few signs, or there may be evidences of a general bronchitis. In these slowly emaciating infants there is no cough of sufficient severity to indicate involvement of the lung. The terminal stage may present cerebral symptoms of a mild type, such as rigidity of the neck, with periods of stupidity alternating with irritability. The infants die with

a progressive loss of flesh and strength. The temperature is for days normal or a little above normal. In other types the disease is masked by an acute or subacute bronchopneumonia. In these cases the infant, after suffering from exposure or some infectious disease, suddenly exhibits all the signs of a bronchopneumonia. There are severe cough, high temperature, dyspnœa, and cyanosis, as in the ordinary bronchopneumonia. Death may ensue in a few days or in a week. In other forms fatal results take place after several weeks, with symptoms closely resembling those of a persistent bronchopneumonia of the ordinary non-tuberculous variety. In other cases the symptoms of an acute bronchopneumonia are present, sometimes complicated with empyema. Evacuation of the pus is followed by apparent improvement, and the empyema may even heal, but the infant or child gradually emaciates, and the cough, which may have abated, becomes aggravated. Examination of the chest reveals new areas of lung involvement. In these cases the pus does not always contain the tubercle bacilli. The empyema may be the result of mixed infection, and the pus may contain only simple streptococci, the physician being frequently misled as to the true condition. Many forms of tuberculosis of the lungs in infants and children cause death with the terminal symptoms of tuberculous meningitis. Especially characteristic in older children, as compared with the adult, are those cases of tuberculosis of the lung which follow some slight injury, blow, or exposure, and in which there are for weeks no signs in the lung or elsewhere to account for the gradual emaciation and intermittent or remittent temperature. After a variable length of time signs of involvement are detected at one apex, or posteriorly over the base or mid-area of the lung. Even then the cough may be absent and no sputum be expectorated. The child then has intervals of stupidity; there is delirium at night accompanied by the typical hydrocephalic cry. Irritability of temper is marked, the emaciation is very rapid, and coma and death with terminal paralyses show that the infection has involved the cerebral meninges.

The TEMPERATURE is irregular in course. It may be normal for a few days, after which it rises one or two degrees daily in the afternoon and falls to the normal toward morning.

HÆMOPTYSIS is very rare in infants. Henoch has seen 3 cases in young infants and 1 in a child of two years. Acker has reported a case in a child of three years. I have seen several cases in children of more than six years of age.

SPUTUM.—Infants do not expectorate. At most a frothy mucus collects around the orifice of the mouth after a coughing spell. Even older children expectorate very little, and must be taught to do so.

Course.—Up to the second year of life, the course of tuberculosis of the lung is generally acute (Henoch). The disease may pursue a subacute course, but it is rarely as prolonged as in the adult. In children above the fifth year its course closely resembles that taken in the adult.

The **diagnosis** of tuberculosis of the lung in infancy and early childhood must, for the most part, be made from the history of the case. In many of the cases the physical signs in no way differ from those seen in non-tuberculous diseases. Cases in which marked consolidation of the lung persists, with progressive emaciation, and cases in which auscultation reveals the presence of cavities, are certainly suspicious. There is no reliable method of determining the nature of an acutely developing bronchopneumonia; the detection of the tubercle bacillus in the vomit, in the feces, or in the exudate of a complicating pleurisy or empyema, is of diagnostic aid.

The existence of enlarged lymph-nodes in the mediastinum or the root of the lung is, according to some authors, revealed by symptoms of pressure. Pressure on the bronchi may give rise to dyspnœa; on the large veins, to nervous congestion and cyanosis, or œdema of the lungs; on the recurrent laryngeal nerves, to asthma or laryngospasm; on the œsophagus to dysphagia. Although in exceptional cases such symptoms may be thus correctly interpreted, I believe with Henoch that diagnosis of these enlarged nodes during life is highly uncertain.

Treatment.—From a study of the symptomatology it will be seen that the treatment of tuberculosis of the lung in young infants and children must be simply symptomatic. A case of suspected tuberculosis should be isolated from other children. The fever needs little attention if it remains low; if high, it is treated as in a case of simple bronchopneumonia. The cough and restlessness are also treated symptomatically.

Tuberculosis of the Peritoneum.

(*Tuberculous Peritonitis.*)

Occurrence.—According to the statistics of Dennig, Müller, Biedert, and Simmonds, tuberculous peritonitis occurs in from 8 to 21 per cent. of all the cases of tuberculous disease. Sixty-five per cent. of the cases operated on by Herzfeld were under the age of fifteen years. The frequency varies in different localities.

Acute tuberculosis of the peritoneum is seen in acute phthisis as a complication, when there may be also an exudate with miliary tuberculosis of the peritoneum. This form of peritoneal tuberculosis is of no clinical interest.

Chronic Form.—This is the form under consideration. It is rare in the newborn; in a statistic of 100 cases Still found the disease most frequent from the second to the fifth year of life. Next in frequency was the period of five to ten years.

Etiology.—Tuberculous peritonitis is rarely if ever primary, although such cases have been described by Henoch and Müller. The peritoneum may become infected through the blood-channels (hæmatogenous); under these conditions tuberculosis of the peritoneum is simply a feature of the manifestation of acute miliary tuberculosis. The peritoneum may become infected through the lymphatics or lymph-channels (lymphogenous). Under these conditions it is the result of infection from adjacent organs, such as the intestines, the genito-urinary tract, the mesenteric, peritoneal, retroperitoneal, or bronchial, lymph-nodes, and the vertebræ and pleura.

Morbid Anatomy.—There are, according to Herzfeld, three main forms of tuberculous peritonitis: the miliary, submiliary or exudative form; the nodular or sclerosing form; and the adhesive form.

The Miliary, Submiliary, and Exudative Form.—In this form there is an eruption on the peritoneal surface, of gray, transparent tubercles of varying sizes. The intestinal coils are covered with fibrin, and are slightly adherent to one another. There is a clear serous, serofibrinous, seropurulent, or even ichorous exudate (mixed infection).

The Nodular or Sclerosing Form.—In this form the quantity of the exudate in the abdominal cavity is small. The omentum is converted into a solid cylindrical mass, containing tumors of a tuberculous nature as large as an apple. The mesentery is thickened and covered with tubercles. The intestinal wall is thickened and covered with gray or grayish-yellow tubercles, which may attain the size of tumors. The coils of gut are adherent, and the whole peritoneal cavity may be obliterated.

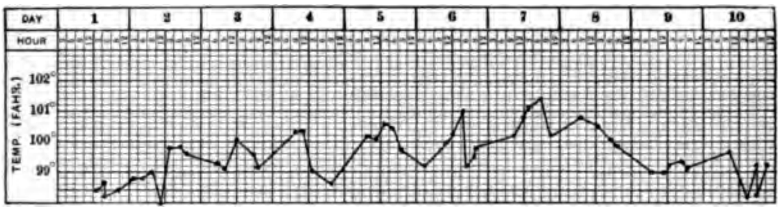
The Adhesive Form.—In this form the intestines form an adherent mass, with masses of exudate between the coils of gut, forming pseudocysts. This exudate may be of a puriform nature. Aggregations of tubercles between the coils of gut break down and perforate into the gut, or become adherent to the abdominal wall and perforate externally forming intestinal or abdominal fistulæ. Perforation may thus occur in the absence of any real ulceration on the mucous membrane of the gut.

In addition to the above principal forms of tuberculous peritonitis, mixed forms occur.

The exudate in the peritoneal cavity may be purely serous (ascites), or the serum may, as in a case which I observed, have a chylous appearance, due to the admixture of fat. In other forms the exudate may be seropurulent, hemorrhagic, or, in mixed infections, putrid. In the purely ascitic variety the fluid is free; in the purulent form, it is frequently sacculated between the adhesions on the coils of gut.

Symptoms.—The disease is, as a rule, insidious and slow in development. The stage of abdominal distention has usually been reached when the patient is first brought to the physician. The history shows that the child has been for some time gradually losing weight, that the appetite is capricious, and that there have been attacks of abdominal pain. This pain may be localized or radiate from one point, may be constant, or may resemble visceral neuralgia. Sometimes there is no history of pain, but it may be detected by pressure on parts of the abdomen. There may be a slight rise of temperature toward evening (Fig. 71); diarrhœa may alternate with constipation. The abdominal distention is the leading feature. It may take the form of a uniform ascitic accumulation (Fig. 72); the surface of the abdomen may be uneven and irregular (Fig. 73), and tumors with cystic formation may be felt through the abdominal walls.

FIG. 71.



Tuberculous peritonitis. Female child, five years of age. Ten days of her temperature immediately preceding operation (laparotomy).

The movements, which are rich in fat, sometimes resemble icteric evacuations. This condition was formerly considered pathognomonic of tuberculous peritonitis (Biedert, Conitzer).

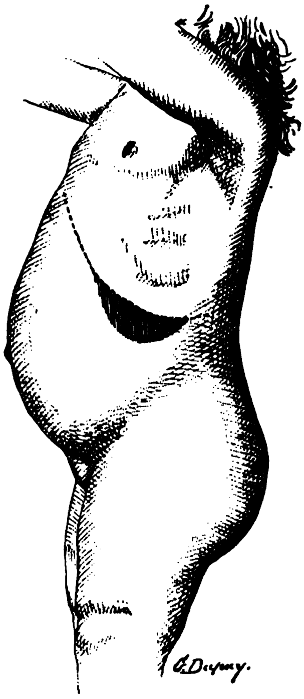
Vomiting of fecal or biliary matter resembling that seen in appendicitis may occur.

In marked contrast with these is a form which in its acute onset may simulate acute perforative peritonitis. In this variety the tubercle mass may cause perforation either of the appendix or the gut in its cavity. Symptoms of acute perforative peritonitis which in every way resemble those of appendicitis set in. It is only by resort to laparotomy that the nature of the affection can be discovered.

Physical Signs.—The physical signs in the miliary and the nodular forms are due to the presence of free fluid in the abdominal cavity. If ascites is present, there will be the percussion-wave, the flatness in the flanks, and change of tympanitic area will occur with change in the position of the patient. If adhesions are present and there are encapsulations of fluid, the signs will not vary on changing the position of the patient. On the other hand, in the adhesive form there will be evidences of tumor masses in the abdominal cavity, cystic formations caused by the encapsulated exudate, and little or no fluid.

In cases of adhesions in tuberculous peritonitis of the miliary form, the fact that when the patient is in the recumbent position the coils of gut may here and there be seen outlined over the abdominal parietes, is of diagnostic value (Fig. 73). I was able by this means to confirm the diagnosis of adhesions in one such case, and have detected them clinically in other cases in which this form of peritonitis had been diagnosed.

FIG. 72.



Uniform abdominal distention due to ascites of tuberculous peritonitis; enlarged spleen.

The liver may be enlarged as a result of amyloid degeneration or tuberculous interstitial hepatitis.

The spleen may be enlarged as a result of amyloid degeneration.

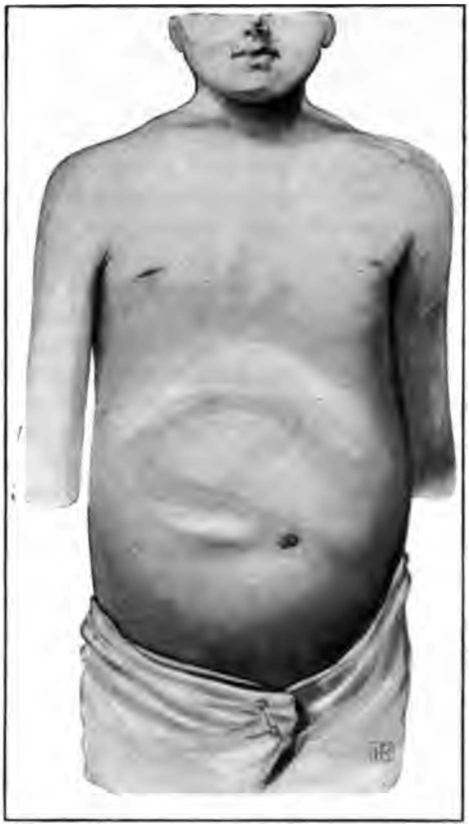
Rectal examination may reveal miliary nodules or peritoneal masses palpable through the walls of the rectum.

The **diagnosis** is based on the slow and insidious onset, the colicky abdominal pains, abdominal tenderness on palpation, the presence of ascites or tumor masses, constipation alternating with diarrhœa, progressive loss of strength, intermittent fever or slight rise of temperature in the evenings, and the presence of tuberculosis in other organs. At the outset tuberculous infection in other parts of the body may be difficult of detection. A rectal examination should always be made. This form of peritonitis should be differentiated from the non-tuberculous form. Inasmuch as some authors, notably Unger and Nothnagel, doubt the occurrence of idiopathic non-tuberculous peritonitis, caution should be exercised in making a diagnosis of simple chronic peritonitis. Absence of emaciation and retrogression of symptoms by no means prove that the disease may not have been tuberculous, since some forms of tuberculosis of the peritoneum present such peculiarities.

This form of peritonitis must also be differentiated from cirrhosis of the liver, new growths, cardiac and renal affections.

In some forms of tuberculous peritonitis, especially of the miliary type, the child will fail to show a temperature above the normal for weeks, and, being in tolerably good condition, the question will arise as to the nature of the abdominal process. In these cases a diagnosis is facilitated by the use of tuberculin. A reaction may be thus attained varying from a degree or more above the normal.

FIG. 73.

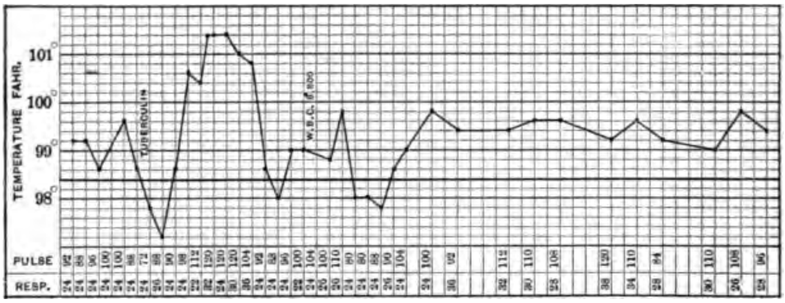


Tuberculous peritonitis, miliary form, female child, five years of age. Irregular contour of abdominal parietes in the recumbent posture, showing intestinal agglutination.

The patient is placed in bed, the temperature previously observed every three hours for a few days, and then is given subcutaneously 0.25 milligramme of tuberculin. If no reaction takes place, 0.50 milligramme is given after a few days. The dose may be increased to a milligramme with older children. A reaction takes place, if the process is tuberculous, within twenty-four hours; though I have seen it delayed for forty-eight hours (Fig. 74).

Course.—The course of the disease is chronic. Frequently the symptoms retrograde and there is an apparent recovery. The ascites may at times diminish, and again increase. The chronic forms unless operated upon lead to the formation of abdominal fistulæ, to perforative peritonitis, to tuberculosis of the organs, and to amyloid degeneration of the liver and spleen, with emaciation, exhaustion, and death.

FIG. 74.



Tuberculin reaction. Miliary form of tuberculous peritonitis. Diagnosis confirmed by operation. Boy four years of age.

Treatment.—Laparotomy, when advanced tuberculosis in other organs is not present, is, according to Herzfeld, curative in 54 per cent. of cases. In a series of 29 cases of all ages operated upon by him, 19 were under the age of fifteen years. With operative treatment must also be combined the medicinal and hygienic treatment suitable to cases of pulmonary or local tuberculosis. On the other hand, in the forms which resemble cases of *tabes meseraica*, in which emaciation and cachexia are present before much exudate is formed, it is difficult to decide as to the propriety of operative measures, especially if diarrhœa be present. In these cases if the diagnosis is not certain, proper feeding should be begun and the condition of the patient improved before laparotomy is attempted.

Other Forms of Tuberculosis.

Tuberculosis of the larynx is rare in children. It occurs in from 3 to 4 per cent. of the total number of cases of tuberculosis (Reiner, Steffen, Barthez, Rilliet). Demme has reported a case in a child of four and one-half years.

Tuberculosis of the Pleura and Pericardium.—Primary tuberculosis of the pleura is rare. Dennig reports that it occurred as a feature of general tuberculosis in 14 per cent. of his cases. Pericarditis of the tuberculous variety occurs in only 3 per cent. of the cases of general tuberculosis.

Tuberculosis of the heart muscle is very uncommon. Sänger reports a case in a child of nine months, and Demme one in a patient of five years. The endocardium may be involved in general tuberculosis (Perroud).

Abdominal Tuberculosis.

The following table shows the relative frequency of tuberculous involvement of the abdominal viscera:

	Peritoneum.	Stomach and intestines.	Mesenteric lymph-nodes.
Dennig	8 per cent.	14 per cent.	21 per cent.
Müller	18 "	38 "	57 "
Biedert	18 "	31 "	40 "
Simmonds	21 "	31 "	53 "

Tuberculous Meningitis.

(*Acute Internal Hydrocephalus; Basilar Meningitis.*)

Occurrence.—Tuberculous meningitis has been observed in infants as early as the third month (Steffen). Barthez and Rilliet have seen cases in infants five months old. The frequency of tuberculous meningitis varies with the locality. Dennig places the frequency of tuberculous meningitis among children who suffer from tuberculous disease at 60 per cent., while Medin found this form of meningitis in 15 per cent. of tuberculous children. It is most frequent in the nursing period; 75 per cent. of all cases occur under the fifth year. The second year of infancy shows the greatest number of cases (Steffen). It is more frequent among male than female children.

Of 26 of my cases of tuberculous meningitis, substantiated either by autopsy or by the presence of tubercle bacilli in the fluid obtained by lumbar puncture, 46 per cent. (12) were under four years of age, 53 per cent. were four years of age or over; the average age was four years and four months. The oldest case was ten years, and the youngest seven months.

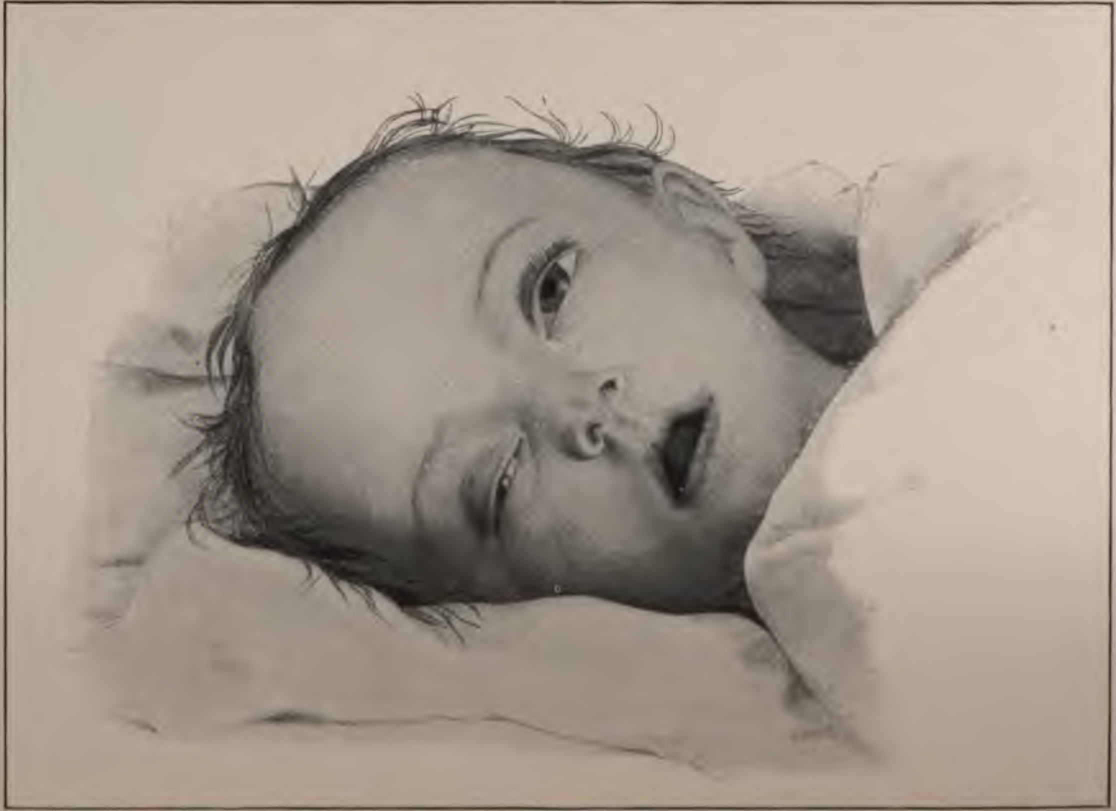
Etiology and Morbid Anatomy.—Exposure to cold and traumatism predispose to the affection. In many cases there is, in addition to the meningeal disease, disseminated tuberculosis of the lungs, pleura, spleen, liver, and peritoneum. In other cases the meninges are the chief seat of the disease, only a few isolated foci of tuberculosis being present elsewhere, as in the mesenteric or bronchial lymph-nodes. It is rare to find the lesions confined to the meninges, and some authors deny the possibility of such a condition. It is not always possible to determine the primary focus of infection.

The tubercle bacilli, which are the causative factors, may be carried by the blood (hæmatogen) to the meninges, and there give rise to a more or less extensive miliary deposit. The original focus in such cases may have been a cheesy lymph-node, a tuberculous nodule in the lung, or a carious bone or joint. The tubercle bacilli may enter the lymph-channels from an adjacent carious bone of the skull, a diseased mastoid, or a spinal vertebra. An ozæna or a solitary tubercle of the brain may give rise to tuberculous meningitis by this mode of transmission. Whatever the focus, at the point of infection there will result an eruption of tubercles which may remain localized or become disseminated along the course of the cerebral lymph-channels. When the dissemination of bacilli occurs through the blood, miliary tubercles are found in the course of the bloodvessels and in their walls. The tubercles may remain confined to the meninges or may involve the brain-tissue, constituting a meningo-encephalitis. In the meshes of the meninges (pia) there is a serofibrinous or seropurulent exudate, which is also found in the ventricles (hydrocephalus), and which infiltrates the brain-tissue itself (meningo-encephalitis). If the exudate is limited in quantity and the tuberculous process is localized, forming one, two, or three nodules, we speak of tuberculosis of the brain or solitary tubercles of the brain. The tubercles are seen as grayish shining nodules surrounded by a hyperæmic zone. They vary from microscopic size to large yellow nodules, which are of older origin. They may be strung along the vessels or break through their walls, forming thrombi with subsequent hemorrhagic infarction.

The basal branches of the artery of the Sylvian fissure are usually the seat of the eruption of these tubercles. The tuberculous process is generally bilateral, but one side, usually the left, may alone be the seat of disease. Tuberculous meningitis affects by selection the base of the brain—hence the term basilar meningitis. The convexity may be involved in the process, although it is rarely the seat of tuberculous meningitis unless the base of the brain is affected. Steffen and Henoch have described cases of isolated tuberculous meningitis of the convexity of one or both sides. The base, the vicinity of the pons, and the chiasm are the seats of the process. In all forms, the pia is infiltrated with serofibrinous or seropurulent exudate. The choroid plexus may be involved. Acute hydrocephalus then results.

The brain-tissue itself is the seat of inflammatory infiltration, and the nerves at the base are surrounded with exudate and are in process of degeneration. The ependyma of the ventricles (the lateral, third and fourth) are the seat of inflammatory thickening and eruption of tubercles. These cavities are filled with exudate. The meninges of the spinal cord, the cord itself, and the nerves at the points of exit are frequently involved in the tuberculous meningitis, as has been demonstrated by Leyden, Erb, and Dennig. The latter found the cord involved in 9 out of 10 cases. The pia may be involved to a slight degree. The nerves at the points of exit are

PLATE XIV.

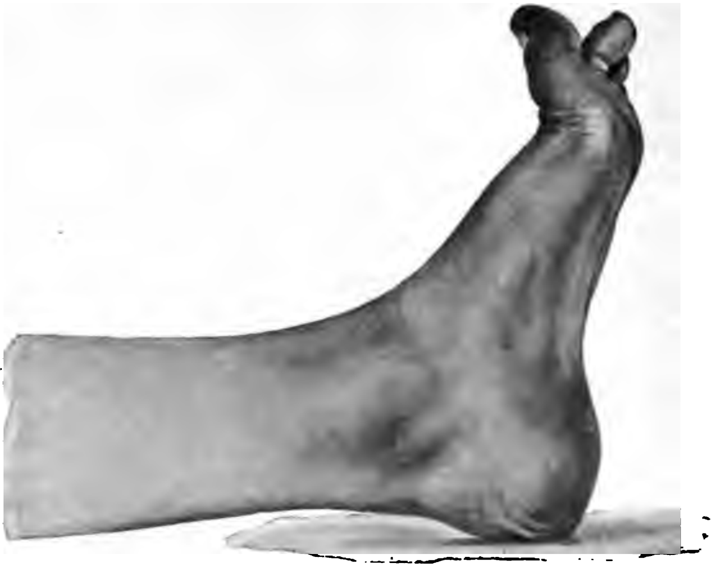


Tuberculous Meningitis. Infant eight months of age. Stage of paralysis, left facial paralysis; left lagophthalmus, bulging fontanelle.

involved in inflammatory exudate. The tissue of the cord and the nerve-elements may be the seat of degenerative processes.

The **symptoms** of tuberculous meningitis cannot be clearly classified according to stages. There is an indefinite period of premonitory symptoms followed rather abruptly by manifestations of cerebral irritation, and ending with a period in which pressure-symptoms are pronounced. As a rule, the disease is slow of development, although cases occur in which the rapid malignant course simulates that seen in rapidly fatal cerebrospinal meningitis of the epidemic type. The disease gives a varying clinical picture in the different periods of childhood. The infant of from seven to twelve months refuses to nurse, has a low fever, and may have diarrhœa alternating with obstinate constipation. The illness of an infant is often attributed to a fall occurring while it was learning to walk. A weakness of the extremities is thus indicated. The infant becomes indifferent to its surroundings and passes into a somnolent condition. Emaciation is progressive. Vomiting occurs once or twice daily, the food being ejected from the mouth after nursing without apparent effort. The vomiting may be followed by a convulsion, after which the infant becomes unconscious. There may be strabismus, or rigidity of the extremities, or the extremities may be in constant motion of an automatic character. The convulsions may follow one another without cessation. These symptoms may set in after a period of one, two, or five weeks of ailing. In other cases the infant may have suffered from a chronic otorrhœa, although otherwise in apparent health. Suddenly, vomiting followed by a convulsion sets in. This convulsion is the forerunner of symptoms, such as coma, which denote that the disease has become established without having attracted the notice of the parents. In children of five years of age the symptoms are more marked. The child may have an attack of vomiting and diarrhœa and apparently recover; after a few weeks, during which there are irritability, loss of appetite, and progressive emaciation, the child no longer desires to be up and about, but lies quiet in its crib, with its head in a characteristic rigid position. It develops strabismus, becomes soporose, and cries out at night. This cry is sometimes piercing in character, and is the cause of much concern to the mother. When the symptoms of cerebral pressure are fully developed, the picture is in the majority of cases much the same. The infant after the first convulsion lies in a soporose or comatose condition. The eyes are open and there is a vacant stare; the sclera may be apparent above the cornea; the fontanelle if still open is tense and bulging, and there may be horizontal nystagmus. The infant cries if disturbed, or may be indifferent to its surroundings. The pupils may be unequal in size and react to light. In one case which I observed the pressure-symptoms were extreme. The infant lay on its back with rigid neck and arched back (opisthotonos), and emitted a piercing cry at intervals. At each cry the pupils became successively dilated and contracted (hippus). I have seen this phenomenon in two cases of tuberculous meningitis. Opisthotonos may be present, and the retraction of the head may relax at intervals, the muscles of the back being lax. In some cases there is apparently no rigidity of the neck. As a rule there are no convulsions. As the infant or child lies quietly in its crib the inspirations during the stage of cerebral pressure may be very irregular or may be of the Cheyne-Stokes type. The outline of the abdomen is at first normal or there may be a slight retraction at the upper part. The abdominal wall may be quite lax, so that the coils of gut can be made out. If the case is protracted, retraction of the abdomen occurs in the final stages of the disease. This condition has been described as the boat-like abdomen. It is not diagnostic of this form of meningitis.

FIG. 75.



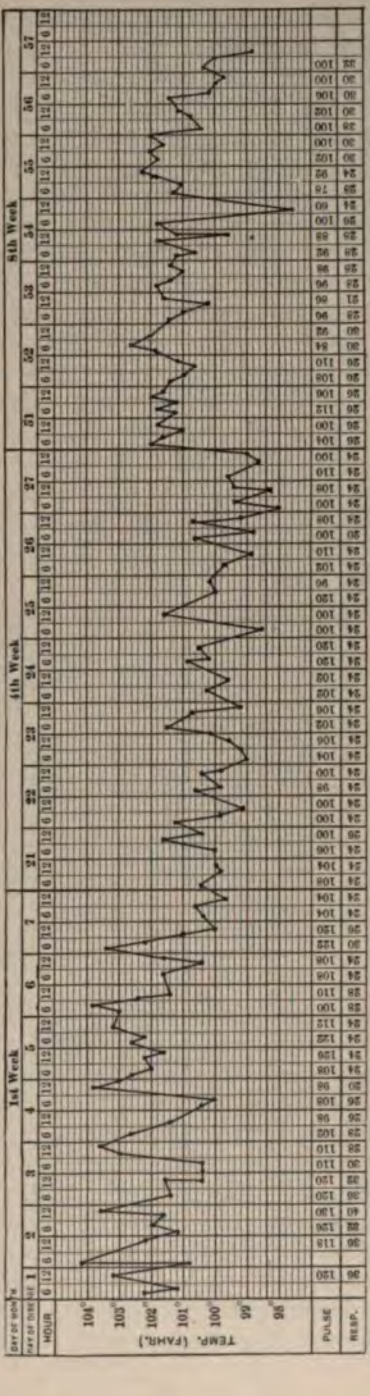
Babinski's reflex. Tuberculous meningitis; stage of facial palsies. Boy, seven years of age.

In rare cases spastic symptoms closely resembling those of tetany occur after the initial convulsion. The infant lies comatose, with rigidly flexed arms; the Chvostek and Trousseau symptoms are present. In all of these cases, if the skin is stroked with the finger ever so lightly, a red mark appears over the stroked area (tache cerebrale). In the spastic cases the knee-reflexes may be increased, but in the non-spastic cases they are diminished. It is difficult to elicit Kernig's symptom in spastic cases, because the infants lie with the knees flexed. By straightening the legs and thighs it is possible in the majority of children to obtain the symptom.

The most important symptoms of the final stage of tuberculous meningitis, both in infants and older children, are the localized facial palsies. For several days or weeks preceding the fatal issue, one side of the face is seen to be flatter than the other. There may be ptosis or lagophthalmus of the eyelids. One eye may be rotated internally, owing to paralysis of the abducens. The extremities are also paretic. The arm and leg of one side may be rigid or flexed, while those of the opposite side are lax. Irritation of the soles of the feet may give a Babinski reaction (Fig. 75). In some cases this reaction is present independently of any irritation of the plantar surface. Toward the end, convulsive twitchings appear in the muscles of one or the other side of the face or of the extremities. Death supervenes in coma with convulsions. The heart may continue to beat for some time after the cessation of respiration.

Children from six to nine years of age present a more decided clinical picture in the premonitory stage. For some weeks before the onset of symptoms of irritation they complain of headaches, frontal, sincipital, or parietal.

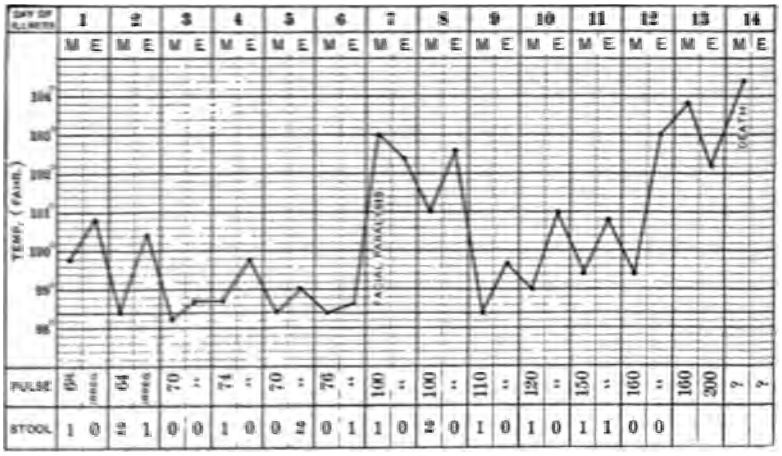
Fig. 76.



Tuberculous meningitis, observed from the outset of the illness. Tuberculous pneumonia over a small area, with dulness; subcrepitant râles at the left apex; no cough; no sputum. First, fourth, and terminal weeks of the illness. Female child, nine years of age.

The patient is listless, walks with an unsteady gait, and has no desire to study or play. A discharge from the ear may have been present for months before the onset of these symptoms. In one case the child had for some time complained of pain in the left side of the chest and had lost weight steadily. There were mild pleurisy and signs of slight consolidation at the apex of the left lung. There was daily elevation of a few degrees of temperature in the evening, and a normal temperature in the morning. In this case, although there were distinct signs of pulmonary involvement of a mild type, the emaciation was progressive and the leucocyte counts low (8000 W.B.C.). At night the typical cry of tuberculous meningitis was present. In the early stages of the disease the patient was conscious during the day, but later became listless, irritable, and slept or was drowsy during the day.

FIG. 77.



Tuberculous meningitis, observed from the outset of the symptoms. Female infant, fourteen months old

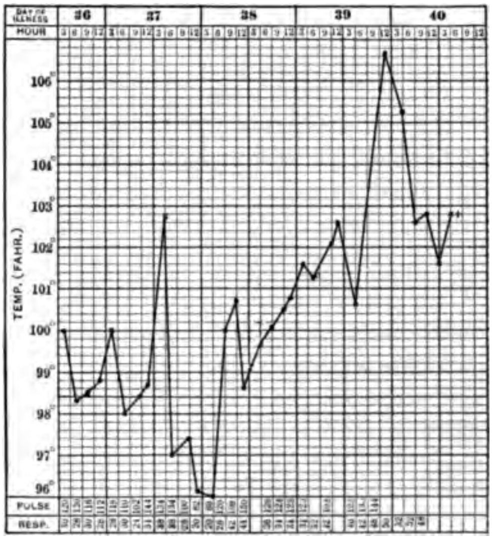
When questioned, a slow stupid answer was given. The child vomited and at times became nauseated. The Kernig symptom appeared. Right lagophthalmos was present. The pupils were unequal in size, the left being dilated. The pulse at this time varied from 60 to 100 and was compressible. Finally, coma set in with left facial palsy and convulsive twitchings of the left side of the face. This case was for three months under constant observation. In other cases the vomiting is rapidly followed by paralytic symptoms such as ptosis and facial paralysis on the same side. There are no convulsions and no cry, but there is rigidity of the neck and extremities; one patellar reflex may be absent. The Kernig symptom and Babinski reflex are present in the majority of cases in children.

The very rapid and fatal cases of tuberculous meningitis have been described by Osler and Dennig. In these the patient is overwhelmed by the toxæmia of the disease, no marked tuberculous lesion being present in any organ but the brain. A patient in apparently good health is suddenly seized with convulsions followed by a period of unconsciousness. There are muscular relaxation and a vacant stare. The convulsions may be repeated at intervals of a few minutes or half an hour. There then follow opisthotonos and spasms, and the abdomen is tympanitic. There is neither vomiting, tache, nor elevation of temperature. There are spastic contractures of the extremities alternating with relaxations. Death occurs in a convulsive seizure within ten hours.

Schlessinger reports a case of tuberculous meningitis in a child two and a half years old, setting in with convulsions, followed by hemiplegia and aphasia within thirty-six hours. After these premonitory phenomena the ordinary symptoms of the disease appeared. Such cases are exceedingly rare.

The temperature-curve in tuberculous meningitis is not characteristic. In some cases the temperature will not rise more than a

FIG. 78.



Tuberculous meningitis; general miliary tuberculosis; terminal stage; coma and paralysis. Boy, seven years of age.

degree or two above the normal, intermitting to the normal or nearly so. In other cases it may be normal for days, then rise a degree or more, rarely above 103° F. (39.4° C.), and then fall again to the normal. In cases in which there is a general miliary process the temperature mounts to 105°–106° F. (40.5°–41.1° C.) or higher toward the close. The fatal issue in other cases occurs with a subnormal temperature (96° F., 35.5° C.) lasting for a day or more before death. If the case is a protracted one, the normal diurnal variations may be reversed—that is to say, the highest temperature may be reached in the morning hours and the lowest toward evening. In the majority of cases, however, the temperature is rarely higher than 103° F. (30.4° C.).

The pulse is increased at the onset, but during the course of the disease becomes slow and may range from 60 to 100 or more during the twenty-four hours.

The respirations are irregular, and may vary from 18 to 60 within the twenty-four hours, even if no pulmonary lesion is present.

Individual Symptoms.

Onset.—Of 26 cases which I have utilized for the purposes of this article, the onset was slow and insidious in 77 per cent. The mother of the child related that the patient was not quite well, or complained of slight headache, and vomited from time to time before the appearance of marked symptoms. In those cases which have come under my observation early in the disease, as early as the second day after marked symptoms were observed by the parents, there was no history of vomiting; as a rule, the child had a slight elevation of temperature, was irritable from time to time, refused to nurse, and on the whole the mother observed a change in the general attitude of the child toward herself and others. It was only in those cases which had lasted at least a week that there was a history of vomiting. It was in exceptional cases, as in one case two years ago, in which the mother asserted that the disease began suddenly with vomiting and convulsions.

Vomiting.—Vomiting sets in, on the average, eighteen days before the fatal issue, and may occur once or twice daily. It may be absent in some cases. With the vomiting there may be localized convulsions, which appear with the vomiting, as has been stated in exceptional cases in which the onset was sudden, or may appear two weeks after the initial vomiting attack.

Rigidity.—There are some cases of tuberculous meningitis in which rigidity of the neck is absent throughout the disease. In only one of my cases was there opisthotonos; and the rigidity, if present, as a rule, was but slightly marked; that is, the head was movable almost to a normal degree. The rigidity is tested simply as the child lies in bed; the head is raised, or an attempt made to draw the chin toward the sternum and note the resistance. In only 25 per cent. of the cases was there palpable rigidity or stiffness of the neck, and this appeared late in the course of the disease.

Hyperæsthesia.—Hyperæsthesia, either of the surface or of the senses, is absent, as a rule, in tuberculous meningitis; that is, the child reacts feebly or not at all to irritation, and would, when roused, momentarily protest and then fall into sopor again. In 90 per cent. of the cases there was an absence of hyperæsthesia either of the surface or of the senses; and in this respect tuberculous meningitis is quite the opposite of cerebrospinal forms of meningitis of the epidemic type, in which hyperæsthesia is the rule and forms part of the general symptomatology of the disease.

Kernig Symptom.—This symptom is present in only 50 per cent. of the cases. It was absent in the others. Its presence or absence does not materially aid in the diagnosis.

Babinski Reflex.—In children over two years of age the Babinski reflex is a valuable guide clinically as to the nature of a meningitis, if meningeal symptoms are present; more so than the Kernig symptom.

Of 26 cases of tuberculous meningitis, the Babinski reflex was present in 15. It is found exceptionally in the cerebrospinal meningitis of the epidemic type, or the suppurative forms of meningitis.

The general reflexes are present in tuberculous meningitis early in the disease; whereas late in the disease, when paralysis supervenes, they are absent.

Pulse.—The irregularity of the pulse is of no special diagnostic value in tuberculous meningitis, and if present is only incidental. The irregularity of the pulse is quite a feature in other forms of meningitis, especially the cerebrospinal type. In these cases the pulse at one moment may be 85, and immediately after may suddenly mount to 120 beats a minute. In tuberculous meningitis, however, the pulse, as a rule, is slower than that of meningitis of the cerebrospinal type.

Respiration.—The respiration is irregular in most forms of meningeal trouble in children. In the tuberculous form of meningitis, after the disease is well inaugurated, the respirations are irregular and shallow, and in a few cases, where cerebral pressure is very marked late in the disease, the respirations may assume the so-called Cheyne-Stokes rhythm. The irregularity of respiration or pulse is scarcely an aid as to differential diagnosis of the form of meningitis present.

Temperature.—Of greater utility in the diagnosis is an exact observation of the course of the temperature. Although there are cases of tuberculous meningitis in which the temperature ranges as high as 104° to 105° F., this high temperature is present only in the presence of complications of the lung, or at a late period of the disease, toward the fatal issue. In most cases of tuberculous meningitis which I have seen, a low range of temperature has been the rule.

Blood.—In all my cases of tuberculous meningitis I have had the blood examined at intervals of three days; in two-thirds of the cases there was a leucocyte count ranging below 20,000 to the cubic millimetre. In the remainder of the cases, however, I obtained a leucocyte count ranging from 20,000 to 25,000 to the cubic millimetre. In some cases there was at some period of the disease a so-called leucopenia. In no case except one, in which the leucocytes just before the fatal issue mounted to 32,000 to the cubic millimetre, did the leucocyte count exceed 25,000 to the cubic millimetre; therefore a leucopenia, however presumptive evidence in the face of other symptoms of the tuberculous form of meningitis, is certainly not a positive evidence of the presence of the disease. The lowest count in my cases was 5000 leucocytes to the cubic millimetre.

Eyes.—The condition of the fundus of the eyes is of special interest in this form of meningitis, as compared with the condition of the disk and retina in other types, such as the cerebrospinal form of meningitis. In 20 consecutive cases of tuberculous meningitis examined by the expert ophthalmologist in my hospital service, the fundus was normal at an early or late period of the disease in 25 per cent. of the cases. In 75 per cent. of the cases, however, there was some change in the disk (optic neuritis), or there were present also tubercles in the choroid. In some cases the disk was simply swollen, and indistinct at the margin; in other cases the veins were congested. Tubercle was found in the choroid in 6 of the 20 cases examined. Choroid tubercle was seen as early as the first and as late as the sixth week of the disease.

The cerebral cry present at night is not distinctive of this form of meningitis; the emaciation, the retraction of the abdomen, the bulging of the fontanelle may be present in other forms of meningitis, especially in that form described by English authors as the posterobasic form. Of great service in making a clinical diagnosis in this disease is the presence of palsies of the cranial nerves, facial paralysis; ptosis, strabismus, paralysis of the internal rectus of one side, or ptosis of one side with or without lagophthalmos of the opposite side, are indicative of a lesion at the base of the brain. These palsies are seen more frequently in the tuberculous forms of meningitis than in the epidemic cerebrospinal type of meningitis. I have, however, seen these palsies in cases of cerebrospinal meningitis either in infants or children, and in these cases the palsies appeared early in the disease rather than late, as in the tuberculous form.

Macewen's Sign.—This sign is elicited by percussion along the parietal or frontal bone over the situation of the anterior horn of the ventricles, and in infants and children below two years of age is of very little value as to the diagnosis of tuberculous meningitis with consequent accumulation of fluid in the ventricle as a result of this disease, inasmuch as in certain children suffering from pronounced rachitis with slight accumulation of fluid in the ventricles, so-called hydrocephalus, this tympanitic note of Macewen may be obtained. The Macewen tympanitic note is therefore of value only in children above two years of age, and must always be sought by sitting the patient upright in bed, inclining the head toward one or the other shoulder, and percussing the inferior side of the skull over the parietal or frontal bone. When carried out in this manner, a marked tympanitic note over the anterior horn of the ventricle is presumptive evidence of fluid in the same as a result of inflammatory processes at the base of the brain and obstruction of the veins of Galen.

Lumbar Puncture.—Lumbar puncture is to-day the most valuable aid we possess in making a positive diagnosis of the various forms of meningitis. In tuberculous meningitis there has been discussion as to the value of an examination of the puncture fluid in the diagnosis. First, as to the cyto diagnosis, it may be said that in 15 of my cases of tuberculous meningitis studied with a view of noting the character of the cell elements in the puncture fluid, 14 showed a predominance of mononuclear cells. In 1 case there was an equal number of mononuclear and polynuclear cells. It would seem, therefore, that in tuberculous meningitis there is a prevalence of mononuclear cells, and that this is so constant that it would appear to be characteristic. There are forms of cerebrospinal meningitis, however, especially the chronic cases, and those of the posterior-basic type of long duration, in which, instead of a polynuclear picture in the sediment of the fluid obtained by lumbar puncture, the mononuclear picture is apt to present itself, thus closely resembling what is seen in tuberculous meningitis.

The *bacteriology* of the fluid obtained from cases of tuberculous meningitis by means of lumbar puncture has been a matter of close study and difference of opinion; whereas Lichtheim, Lenhartz, and Bernheim found that tubercle bacilli were constant in the sediment of the fluid obtained from these cases; Cassell and Marfan have asserted that their presence is only occasional. Of late we have examined the puncture fluid of 14 consecutive cases of tuberculous meningitis, which were clinically diagnosed as tuberculous in character before the puncture. In 13 of these cases tubercle bacilli were found. The fluids were carefully centrifuged, and the search in some cases was exceedingly painstaking. In some cases, especially of children coming under observation late in the disease, tubercle bacilli were not found during life in the puncture fluid, but were found post mortem. This is explained by the fact that in these cases the tubercle bacilli were present in but few numbers which during life were kept evenly distributed throughout the subarachnoid space, and were found in the puncture fluid only after prolonged search. I am inclined to believe that the search for tubercle bacilli in the puncture fluid obtained from cases of tuberculous meningitis is the most positive and valuable aid to the diagnosis, and the bacilli can be found in the majority of cases, if carefully looked for.

Differential Diagnosis.—Tuberculous meningitis must be differentiated from epidemic cerebrospinal meningitis or sporadic cerebrospinal meningitis, suppurative forms of meningitis, posterior-basic meningitis, apex pneumonia, typhoid fever, sepsis, disturbances of the stomach and gut, uræmia, helminthiasis, and finally the various forms of otitis. From cerebrospinal meningitis it can be differentiated by the slow onset, by the absence of opisthotonos, and in the majority of cases a slight rigidity of the neck, by the absence of hyperæsthesia, the presence of changes in the fundus of the eye, either optic neuritis or the presence of choroid tubercle, which will be absent in cases of cerebrospinal meningitis and posterior-basic meningitis, by the low range of the temperature, by the absence of a leucocytosis above 25,000 to the cubic millimetre, and finally by the results of an examination of the fluid obtained by lumbar puncture.

Pneumonia with cerebral symptoms may simulate tuberculous meningitis. Here again the history and the character of the delirium in older patients will aid us. The signs in the lung and the presence of leucocytosis, which is marked in pneumonia and generally absent in tuberculous disease, are significant. In the majority of cases of typhoid fever the history will be of service in connection with the roseola, the Widal reaction, the enlarged spleen, and the absence of leucocytosis. Diarrhœa may be present in typhoid.

Disturbances of the gut, uræmia, and helminthiasis may present symptoms resembling those of tuberculous meningitis, but the symptoms in time retrograde or are cleared up by a study of the case.

I have seen otitis media in nurslings with very limited areas of bronchopneumonia, simulate tuberculous meningitis. In these cases the infants may have been ill for two weeks or more. They start from sleep, are irritable on awakening, and lose appetite.

In one case the ocular symptoms closely simulated those of tuberculous meningitis. As a rule there are intervals during which the child is not only free from pain, but also has a normal temperature. At other times the temperature has a septic intermittent character, and mounts higher (104° F., 40° C.) than in tuberculous meningitis. Aural examination only will remove doubt.

The **duration** of the disease varies within wide limits; I have seen cases which extended over three months. The majority of cases last from two to three weeks, but cases lasting five weeks are not unusual. The very rapid cases in which death ensued within twenty-four hours have been mentioned.

The **prognosis** is always fatal. Isolated cases of recovery have been reported, but should be regarded with doubt.

The **treatment** is directed to alleviating the sufferings of the patient. Lumbar puncture is not curative, and should not be repeated after the first diagnostic puncture has been performed.

Tuberculosis of the Brain.

(*Solitary Tubercle of the Brain.*)

In this there may be a single localized tuberculous nodule or mass in the brain, or several such formations may be present. Demme found a growth of this kind in an infant twenty-three days old. Henoch has published a case in an infant eleven days old. The majority of cases occur between the second and the fifth year.

Morbid Anatomy.—Tubercle bacilli of diminished virulence and limited number are carried from the focus of tuberculosis to the brain through the blood-channels, and there lodged in a terminal bloodvessel, forming solitary tuberculous masses varying from the size of a pea to that of a hazelnut. These are surrounded by a zone of granulation-tissue. The neuroglia in the immediate vicinity is the seat of proliferation, and may form a capsule around the growth. Circumscribed meningitis over the situation of the growth, with adhesions of the pia mater to the dura, may be present. Fully half of these solitary growths occur in the cerebellum (Gerhardt). The growth may be single or there may be one large growth and several of smaller size. Starr and Seidl found a solitary growth in 77 per cent. of the cases. The larger number of brain tumors in infancy and children are tuberculous. Starr found this variety in 152 out of 300 cases of all kinds of tumors.

The symptoms are those common to all tumors, and will be described in the section devoted to Brain Tumors.

XVIII. SYPHILIS.

Acquired Syphilis of Infancy and Childhood.

Etiology.—Of 42 cases of acquired syphilis collected by Fournier, 19 were infected by the father or mother after birth, and 8 by the nurse. No case was infected in passing through the maternal parts, and no infant was infected by the mother if she had contracted the disease just prior to her accouchement. A child of a syphilitic mother, if born free from signs of syphilis, cannot contract a primary lesion at birth from the maternal parts, even if these parts are the seat of condylomata, nor can such an infant be infected subsequent to birth. It has an acquired immunity against the disease.

A chancre or primary lesion is, in the infant as in the adult, the only evidence of acquired syphilis. It is the result of infection, and must be present in order that the diagnosis may be certain. Chancres are rarely genital. They are found, as a rule, in the mouth, on the face, and on the abdomen and perineum. An infant may be infected by the nipple of the nurse's breast. The act of kissing, contaminated nipples of the nursing-bottle, instruments, sponges, ritual circumcision, and humanized vaccine virus, are all means of infecting the infant. Since humanized vaccine virus is no longer used, this mode of infection has been eliminated.

The **symptoms** consist of a chancre or initial lesion, rarely genital, which appears three or four weeks after inoculation. The other accidents, such as bubo or adenopathies, the eruption, and all the secondary symptoms of acquired syphilis, appear in due course as in the adult. The genital chancre is seen in infections caused by ritual circumcision.

The **prognosis** as to life is good in comparison with that in the hereditary form of the disease. While in the hereditary form the mortality is from 70 to 80 per cent., that in the acquired form is very low. Fournier lost only 1 in 42 cases of acquired syphilis. The course in infants and children is benign. The chancre is not well developed; the induration is present only a short time, or may even escape notice. The infants enjoy good health in spite of the presence of the secondary symptoms. I have confirmed these statements by observing 7 cases of genital chancre. The tertiary manifestations, such as gummata, bone lesions, joint-affections, eye and laryngeal symptoms, and cerebrospinal lesions, appear from five to twenty-five years after the initial lesion.

Differential Diagnosis.—Acquired syphilis must be differentiated from the hereditary form of the disease. Hereditary or congenital syphilis appears early without an initial lesion, showing general secondary symptoms from four to six weeks after birth. The chancre is the first manifestation in acquired syphilis. In Fournier's 42 cases the chancre appeared during the first year of life in 19, and during the second year in 10 cases. The snuffles, pemphigus, and pseudoparalysis are not present in acquired syphilis. Secondary accidents, such as mucous patches or papules about the genitals, appearing during later childhood are probably traceable to a postnatal infection. Interstitial keratitis, bone syphilis, and cutaneous stigmata are common to the hereditary and acquired forms of the disease. It is sometimes very difficult to decide which form of the disease is present. Thus far no one has shown conclusively that Hutchinson's teeth are present in acquired forms of syphilis in infancy and childhood. Their presence is therefore strong presumptive evidence of hereditary syphilis.

Late Hereditary Syphilis.

(*Syphilis Hereditaria Tarda.*)

Fournier defines late hereditary syphilis as a symptom-complex of accidents of syphilis originating in a hereditary infection, which manifests itself at a more or less advanced period of life, that is to say, in the majority of cases between the third and the twenty-eighth year. There are two classes of cases. In the first, the patient has remained in perfect health without any of the eruptive or other symptoms of hereditary syphilis until at an advanced period of childhood one or more of the symptoms of late hereditary syphilis are developed. In the second, the late symptoms have been preceded by the early symptoms of hereditary syphilis. The late symptoms may develop after an interval of from ten to fifteen years. The cases of the former class have been the subject of much discussion. The occurrence of the second class of cases is now well established; it is often very difficult to determine the hereditary or acquired nature of the original infection.

Fournier, in classifying the symptoms of 212 cases of late hereditary syphilis, found the eye to be the organ most frequently affected. Next in order of frequency are the lesions of the bones and skin. The rarer affections are those of the kidney, larynx, spinal cord, testes, and lungs.

The subjects of late hereditary syphilis have certain well-defined general characteristics. They are constitutionally delicate and have an emaciated habitus. The skin presents a grayish anæmia. There is an arrest in the development of bone and musculature. The men are undersized and present the picture which has been characterized as infantilism. The signs of virility, such as the beard, hair under the arm and on the pubes, are scantily developed. The testes are rudimentary. The adult has the appearance of a boy of fourteen or fifteen years. The women are correspondingly backward in development.

The Eye.—The eye symptoms appear most frequently at the age of ten or fifteen years, but may become evident as early as the third year. The principal symptom is a keratitis of the diffuse interstitial variety, the so-called keratitis of Hutchinson. The cornea has a slightly cloudy or filmy appearance, or the whole structure is diffusely opaque. The other ocular accidents are plastic iritis, which fixes the iris, thus limiting its action and causing a difference in the size of the pupils. The rarest manifestations are miliary gummata of the iris.

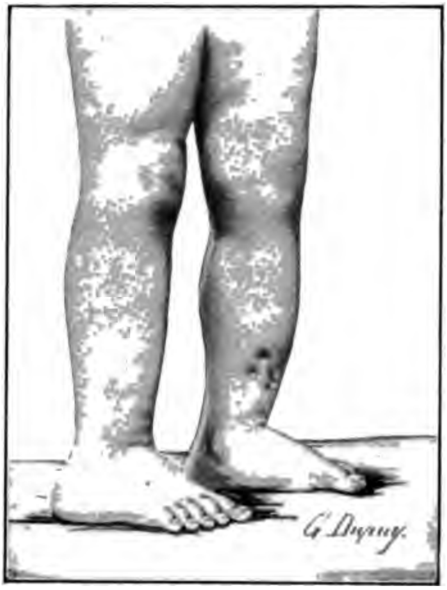
The **bone-lesions** are most frequent between the fifth and the twelfth year.

The *head* presents a cuboidal shape; the forehead is prominent;

the frontal bones have large bosses, as have also the parietal bones. The longitudinal suture is depressed, giving a natiform shape to the head. The cranium may have the form seen in mild degrees of hydrocephalus.

The *nose*, on account of the destruction of the bony septum, has a depressed bridge. The bony and cartilaginous septa form an acute angle, and a peculiar *retroussé* appearance is given to the organ. Both bony and cartilaginous septa may be destroyed. The whole organ is flattened, the tip of the nose being wrinkled into three or more folds.

FIG. 79.



Late hereditary syphilis; bone deformity and sinus. Child, three years of age.

The *long bones* are especially affected by the accidents of late hereditary syphilis, the tibia being most frequently affected. The lesion may consist in an osteoperiostitis, a gummatous osteoperiostitis, or a gummatous osteomyelitis.

If osteoperiostitis is present, there are diffuse swelling and thickening of the bone—the so-called sabre-like deformity (Fig. 79.) This process may affect the long bones of the upper extremities. The gummatous lesions of osteoperiostitis form numerous irregular painful swellings on the bone. Gummata are present on the flat bones of the cranium. When these break down, the destructive processes may expose the dura mater. Arthropathies with synovitis may be mistaken for tuberculosis of the joint. This form of synovitis is generally bilateral. One of my cases, a child five years of age, gave no history of syphilis. The radius on both sides was affected by osteoperiostitis. The joints may be deformed by osteophytic growths involving the epiphysis or head of the bone.

The **ear** is affected by an otitis with destruction of the ossicles, and even by mastoid disease. In other cases deafness supervenes without premonitory symptoms.

The **skin** and **mucous membranes** show certain stigmata in the form of cicatrices of recent or old ulcerations. These may exist on any part of the body, but are especially characteristic on the vermilion border of the lips and at the corners of the mouth, where they are seen as radiating, linear pale-white fissures.

The **lymph-nodes** may be enlarged, especially those on each side of the neck, below the jaw, and in the axilla and inguinal regions.

The **spleen** is enlarged, but not so frequently as is stated by some authors. Fournier found it enlarged in 15 out of 212 cases.

The **liver** was enlarged in 25 cases. In one of my cases of late hereditary syphilis in a child eight years of age, post-mortem examination revealed cirrhosis of the liver of the hypertrophic type. There were enlargement of the spleen, icterus, and ascites; Hutchinson's teeth were well marked, and there were also adenopathies and vasculitis.

Fournier among others has described forms of idiocy and epilepsy of syphilitic origin, but there is great difference of opinion on this question. The theory of Parrot, that rachitis is the result of syphilis, is now generally abandoned. The deformities of the teeth which occur in late hereditary syphilis will be found fully described in the section devoted to Dentition.

Congenital or Hereditary Syphilis.

Congenital or hereditary syphilis results from the infection of the ovule or fœtus in utero. This may occur in a number of ways, but in the great majority of instances it results from infection of the fœtus through the father. The more recent the syphilis of the father, the more likely is the infection to occur. It is most certain to occur if both the father and mother suffer from recent syphilis at the time of conception. The father may at the time of insemination suffer from recent syphilis and the mother be healthy. Under such conditions the child is born syphilitic. The mother may not show any signs of active syphilis either during pregnancy or at any subsequent period. The mother may suckle her offspring, which shows all the marks of active hereditary syphilis, without becoming infected, but the child will infect any strange nurse. The mother has during pregnancy acquired an immunity against the infection. This phenomenon, which is a matter of daily observation, was first brought to the notice of the profession by the distinguished surgeon Colles, and has since become known as Colles's law. The longer the mother is subjected to the influence of the syphilitic virus, the more permanent does her immunity become. Thus a mother who has at first miscarried may eventually give birth to a living infant which bears the marks of syphilis. As the virus becomes weakened, the mother may bear an infant to all appearances healthy. In the interval, although repeatedly pregnant, the mother has shown no signs of active syphilis.

If the father is healthy at the time of insemination and the mother the subject of recent syphilis, the infant will be born syphilitic. On the other hand, if the mother contracts syphilis after conception, the father at the time of conception having been healthy, the infant may or may not be born syphilitic. The nearer the time of the infection of the mother to the end of her period of pregnancy, the more likely is the infant to escape (Monti, Zeissel, Hutchinson). Such an infant if born healthy may become infected in the ordinary way from the mother after birth.

A father who has passed through the secondary manifestations of syphilis may in the late secondary period or tertiary stage fail to convey the poison in the sperma. The result will be an infant free from syphilis (Fournier, Neuman). Yet so far-reaching is the influence of the syphilitic dyscrasia that such an infant, although born healthy and at no time showing signs of syphilis, may present certain signs, such as peculiarities of bone formation (teeth) traceable to the syphilitic virus (parasyphilitic).

Exceptions to Colles's law occur, as is to be expected. Fournier has recorded cases in which mothers apparently immune have developed signs of secondary syphilis after the birth of the infant. Finger has met cases in which tertiary syphilis developed in the mother subsequent to pregnancy without the occurrence in her of any of the signs of secondary syphilis.

Of 218 mothers who had borne syphilitic infants, Hochsinger found 72 who were free from manifestations of secondary or tertiary syphilis although observed for years.

Morbid Anatomy.—In considering the pathology of hereditary syphilis, Hochsinger divides the cases into four classes:

The first class of cases die in utero before the eighth month, Autopsies upon such fœtuses show general parenchymatous involvement of the glandular apparatus with epiphyseal osteochondritis.

The second class includes infants born living or dead before the end of pregnancy. They present at birth a papulobullous syphilide. In these cases diffuse parenchymatous changes are found in the viscera, and frequently marked epiphysitis.

The third class comprises infants born living and without any exanthema, but which later develop an exanthema independently of visceral or bony changes.

The fourth class comprises infants born without an exanthema, but having at birth marked visceral and bone-changes.

Taking up in detail the lesions found in the various parts of the body, we find that the **skin** shows an increase in the thickness of the rete Malpighii, caused by swelling of the cells of the rete, serous infiltration of this layer, and an increase of the spaces between the cells of the rete. The horny layer of the skin is much thinned in comparison, although there is a constant throwing-off of the cells of this layer in lamellæ. The epithelium of the sweat-glands is swollen and there is a small round-cell infiltration between the glands. There is a vasculitis of the small bloodvessels affecting the external coat chiefly. Pemphigus and bullæ result from infiltration of the rete and the lifting up and separation of the horny from the papillary layer by serum.

The Lungs.—The changes in the lungs may be considered under two heads:

First, the lungs of infants born dead or who have died soon after birth, are collapsed, devoid of air, hyperæmic, and dark red in color. In rare cases the lungs may be diffusely whitish yellow in color, giving the appearance of the so-called pneumonia alba. The second class comprises infants that have breathed, and that show a gray or grayish-white discoloration of the lungs in places. There is residual air in the lungs, and they are denser and larger than is normal.

Ziegler has shown that the changes in the lungs consist chiefly in an increase in the interalveolar connective tissue, the formation of new vessels, and vasculitis of the bloodvessels. In the majority of newly born infants the alveolar epithelium is but little affected. In pneumonia alba there is a proliferation of the alveolar epithelium, giving a peculiar appearance and color, hence the name.

The Liver.—Changes in the liver are quite constant in hereditary syphilis. These may or may not be associated with enlargement of the organ. Out of 148 cases of congenital syphilis, Hochsinger found the liver enlarged in 46; in all but 2 the spleen also was enlarged; in the severer cases the liver was markedly enlarged.

The pathological changes in the liver have been described by Hudelo, Hochsinger, and Heller. There may be simply diffuse, small round-cell infiltration of the interstitial connective tissue, with inflammatory changes in the smaller arteries. The liver in these cases is not enlarged. In the cases presenting an enlarged liver there is interacinous proliferation of connective tissue, beginning at the periportal region and following the course of the bloodvessels. There is vasculitis, shown in a thickening of the adventitia of the bloodvessels. The parenchyma is degenerated. In other cases interacinous collections of small round cells are on gross sections of the liver seen as yellow pinhead-sized spots. These are called by Hochsinger miliary gummata. Fully developed gummata of large size are very rare in the liver of infants affected with hereditary syphilis.

The **spleen** is in some cases enlarged to ten times its normal size. Gummata, single or multiple, occur, but are rare. In hereditary syphilis not only is the parenchyma increased, but also the connective tissue of the spleen.

Kidneys.—In rare cases there are induration and contraction of the kidney. The parenchyma is retarded in development by intra-uterine syphilis and the connective tissue increased.

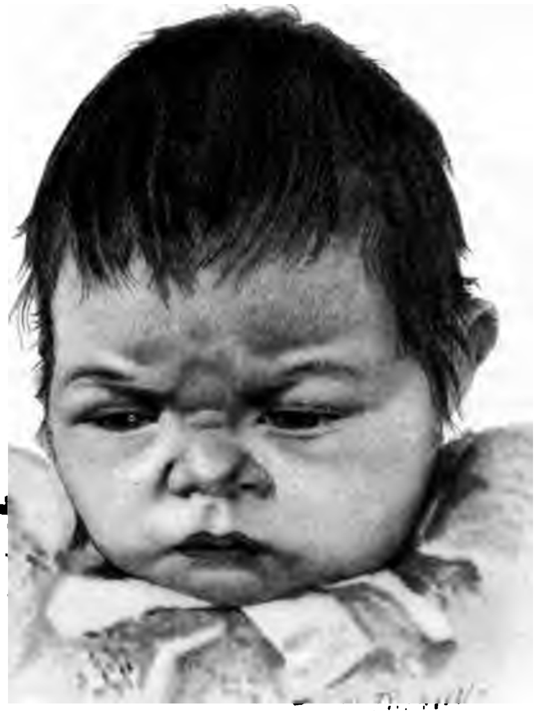
The **pancreas** may be enlarged and infiltrated, the parenchyma hard, and the interstitial connnective tissue increased. There may be condylomatous ulcerations on the tongue, pharynx, and tonsil.

According to Hochsinger, the **glandular apparatus** of the gut may show a diffuse small-cell infiltration, Peyer's patches may be infiltrated, and the vessels may be the seat of a vasculitis. The lymph-nodes are, as a rule, little changed except in cases with late manifestations. The thymus gland in cases of hereditary syphilis has been found to be the seat of cystic degeneration (Eberle, Ribbert), caused by the dilated epithelial spaces of the fœtal thymus.

Bone-changes.—The bone-changes in hereditary syphilis occur principally at that part of the bone between the epiphysis and diaphysis in the lower end of the femur, tibia, and radius. In the milder forms of bone-change there is, according to Ziegler, little real inflammation. There are irregularity in the deposit of lime salts and the formation of marrow-spaces. In severe forms there is a true inflammatory process. In the vicinity of the joint-cartilage, grayish-red, yellowish-white, or yellowish-green foci of osteomyelitis are found. The irregular deposit of lime salts and the formation of marrow-spaces are evidenced by reddish-yellow projections of marrow-spaces into the adjacent proliferated cartilage. These give the epiphyseal junction a more irregular and widened appearance than is normal. Sometimes separation of the epiphysis at the junction of the diaphysis occurs. The above changes are frequent, although not constant. In the later stages of syphilis in children there are, as in the adult, caries, necrosis, and gumma formations in the long and flat cranial bones.

Symptoms.—The symptomatology of hereditary syphilis varies largely with the class of cases. In some cases the fœtus is expelled dead, bearing the marks of fully developed syphilis in the shape of skin, bone, and visceral lesions. In others the infant is born living, but presents a few very characteristic signs of syphilis, such as the presence of bullæ or pemphigus either on the palms or on the soles of the feet. The vesicles may be filled with a purulent fluid. As a rule these infants are emaciated. In some cases the bridge of the nose is sharply depressed and forms a distinct angle with the cartilaginous septum (Plate XV.). This intra-uterine deformity in the newborn infant has been studied by Epstein. Such infants suffer

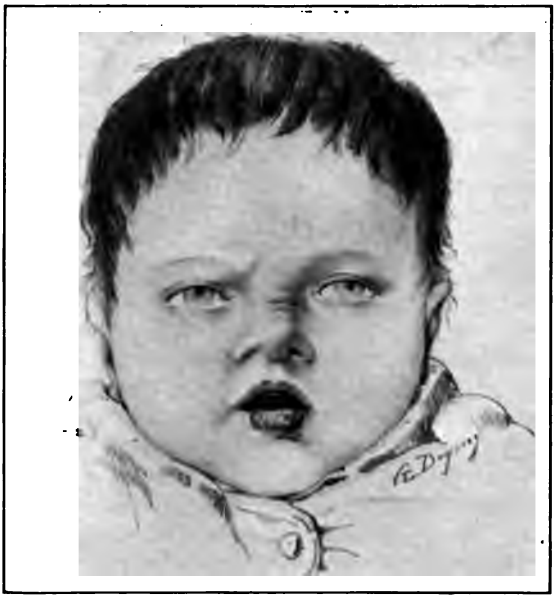
PLATE XV.



Congenital Syphilis. Showing nasal deformity.
Newborn infant.

from a troublesome coryza and cannot breathe freely through the nose. They present enlargement of the liver and spleen, and there may be a few copper-colored discolorations on the skin of the forehead and nose. The lips have a shiny, glossy appearance, and after a time may present distinct rhagades. Some days after birth there is a diffuse syphilitic eruption of papules or vesicopapules, with the so-called diffuse induration of the skin of the palms of the hand and soles of the feet, described by Hochsinger. Here and there discolored spots which were formerly mistaken for papules may be seen. The skin of the face may have a diffuse coppery color. Patches of discolored skin appear and become confluent, the coryza and rhagades along the lips and at the angle of the mouth become more marked, and the rhagades bleed easily.

FIG. 80.

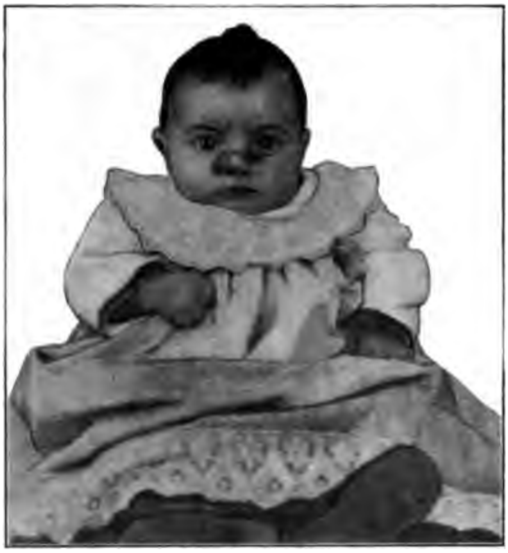


Hereditary syphilis: rhagades and mucous patches of the lips.

In another class of cases the infant is born well nourished and has a good color. Within from two to four weeks a general eruption of papules and vesico-papules appears. Some of the vesico-papules are purulent, and after bursting dry up, leaving the surface covered with crusts on a copper-colored base. In these cases the manifestations on the mucous membranes, including coryza, mucous patches, and rhagades are also gradually developed (Fig. 80). If the above symptoms are marked, we may find enlargement of the liver and spleen. I have seen the most marked signs of hereditary syphilis of the skin without the slightest enlargement of the liver or spleen. As a rule the arms will present papules, which may ulcerate at the points of contact with adjacent surfaces of skin. The typical condyloma lata is not frequent in early hereditary syphilis. The nates have a coppery shining color, are cracked in places and diffusely indurated (Hochsinger's induration). The trunk may present few symptoms. The bicipital glands are enlarged if the syphilitic exanthema is fully developed. The thighs show brownish, copper-colored patches. These patches give the skin a marbled appearance, which differs from that of the so-called healthy marbled skin in that the discolored areas are surrounded by normally colored skin, while in ordinary marbled skin the opposite condition obtains. On exposed areas, such as the knees, nates, soles of the feet, and palms of the hands, the skin is diffusely indurated.

In a detailed consideration of the lesions, those of the skin are the first to engage attention. The most common forms of eruption are the papular or the papulopustular form of syphilide. This may be combined with the macular form; in fact, it is common to find in the same case all forms in various stages of development.

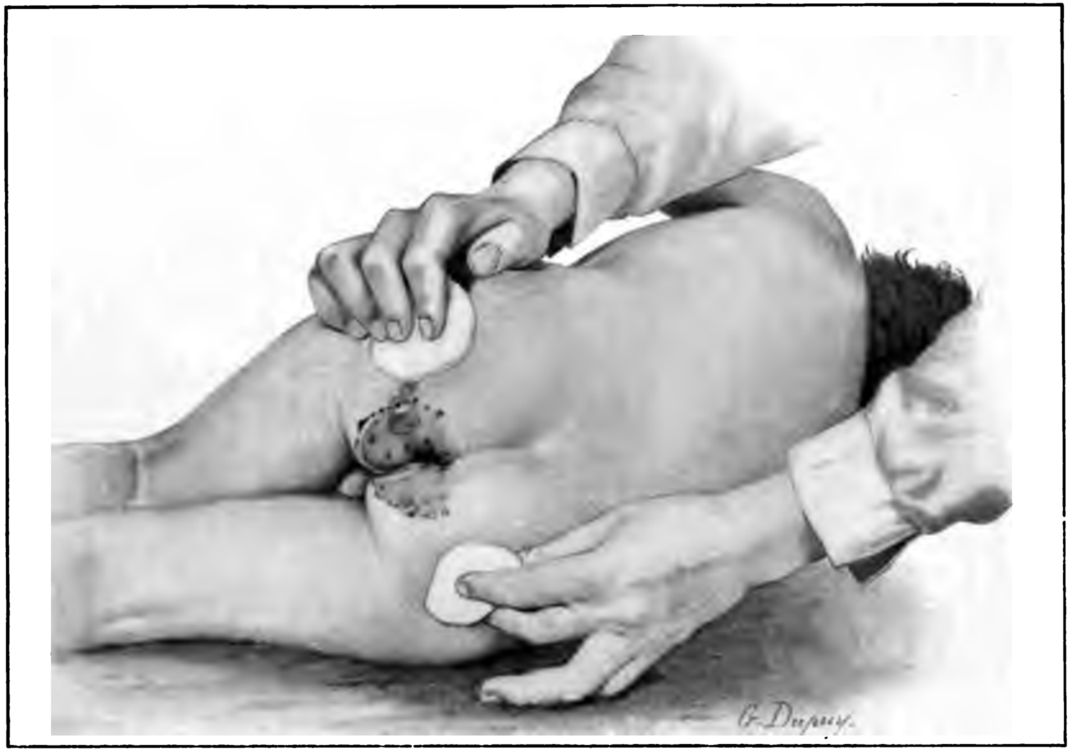
FIG. 81.



Congenital syphilis: circinate syphilide of the nose.

The papules occur on the forehead, palmar surface of the hands and plantar surface of the feet, and on the nates (Fig. 81). They show a distinct induration of the skin, are raised above the surface, and have a glossy, copper-colored appearance. On the nates or in the groin the papules may ulcerate; very rarely these form condylomata

PLATE XVI.



Mucous Patches or Flat Condylomata in a Child Twenty Months of Age. Congenital syphilis.

lata in the early periods of congenital syphilis. The condyloma is a feature of the later period of this disease. Macules develop within the first three months of life, and from the sixth to the tenth week are associated with seborrhœa. Infants thus affected are born with a peculiar anæmia, in which the skin has a cadaveric hue. The macules appear on the forehead and face as copper-hued spots, which increase in number until the skin has a general marbled appearance (roseola syphilitica). They then fade, leaving the surface covered with brownish-red areas. These persist around the alæ nasi and the forehead for a long time, giving the face a peculiar dirty-yellow spotted appearance.

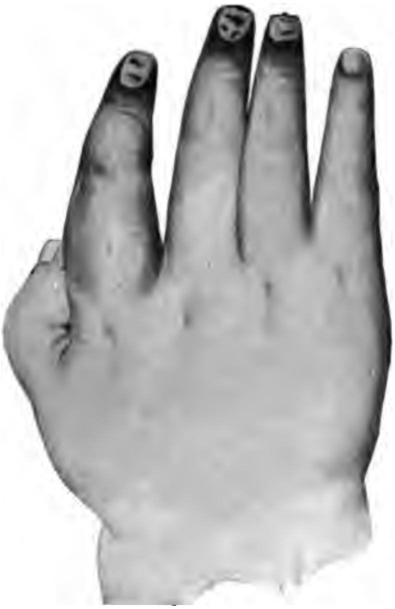
The diffuse syphilitic infiltration of the skin has been studied by Hochsinger. It is not the forerunner or the sequence of any papular eruption. It appears in the third week in 50 per cent. of the cases, and reaches its height between the eighth and the tenth week. It first presents discolored areas on the palms and on the soles of the feet, on the nates, the calves of the legs, also on the cheeks and chin, where it forms rose-colored or copper-colored areas which coalesce. The soles and palms may appear diffusely red or bluish and glossy. The skin is diffusely thickened on the palms and soles and desquamates in lamellæ. At the junction of the mucous membranes and skin fissures result on account of the thickening of the skin. The lips appear anæmic as a result of the infiltration of the mucous membrane, and are fissured. There are rhagades at the alæ nasi. The rhagades at the angles of the mouth are covered with a bluish-white pellicle, and the surrounding skin is copper-colored. There are swelling of the nasal mucous membrane and a thin, purulent discharge mixed with blood. The hair falls out on account of the infiltration of the scalp; the scrotum is thickened and fissured from the same cause.

The blood shows all stages of anæmia, from the mildest to the grave pseudoleukæmic anæmia of von Jaksch, which some authors trace to syphilitic influences.

The bones are affected with an osteochondritis, already described. This may appear in the first few weeks or at a much later period. It manifests itself by pain in moving the joints. The infant cries when handled. The mother notices that one arm lies motionless at the side, and that every attempt to move it causes pain. Parrot described this condition as a pseudoparalysis. At the junction of the epiphysis and diaphysis at the lower end of the humerus or radius the bone may be swollen and painful. As a rule, the process affects the upper extremity on one side only, but in severe cases both the upper and lower extremities may be involved. In some cases this symptom may be present without a skin eruption. The other conditions which simulate it are septic osteomyelitis involving the joints, scurvy, and severe rachitis. I have known instances in which prolonged observation was necessary to clear up the case.

A very characteristic but not common affection of the bones is the so-called dactylitis syphilitica (Fig. 82). This may appear as early as the fourth week, and may be associated with swelling of the epiphyses of the long bones. It consists of a fusiform swelling of the phalanges of one or more fingers. According to Taylor, this is primarily a gummatous infiltration of the skin, the periosteum, bone, and epiphyseal cartilage. In another form the periosteum and the bone itself are the seat of the gummatous inflammation, the epiphysis and the joint becoming involved later in the process. In neglected cases, fistulæ and destruction of the joint may result from necrosis of the epiphysis. The diagnosis of these forms of dactylitis from tuberculous spina ventosa is sometimes difficult, and often impossible without mercurial treatment. Cases of rachitis which involve the phalanges of all the fingers simulate very closely the above affection (see Rachitis).

FIG. 82.



Congential syphilis: onychia of all the nails; dactylitis of the phalanx of the index finger. Infant, four months of age.

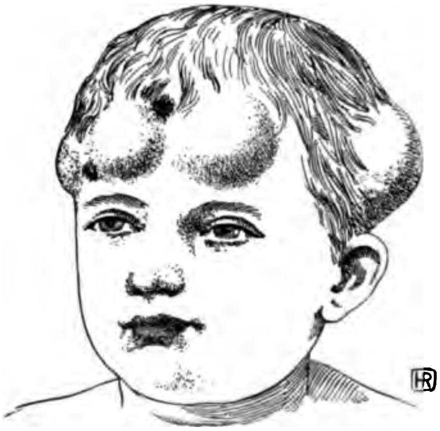
Syphilitic affection of the liver gives no symptoms. Henoch records cases in which icterus was associated with enlargement of the organ. Hochsinger denies the occurrence during the nursing period of any authentic case of syphilis of the liver with icterus or ascites.

Somma, Fischl, and Kohts have described symptoms of cerebral syphilis in infants that were subjects of hereditary syphilis. Convulsions, hydrocephalus, epilepsy, and paralyses have been traced to the presence of gummous meningitis or sclerosis. That such changes occur as a direct result of syphilis at so early a period is doubted by Henoch. I have not seen manifestations of cerebral syphilis in infants. Henoch is also inclined to include Mracek's cases of hemorrhagic syphilis among the septic diseases of the newborn occurring in syphilitic infants.

Antonelli in 1897 described changes in the fundus oculi of newborn syphilitic infants. These consisted of optic neuritis, retinitis vascularis, and retinochoroiditis. He believes these changes to be causative in the production of myopia and strabismus in such infants.

The **diagnosis** of hereditary syphilis is not difficult in the vast majority of cases. If the fœtus is expelled dead, it bears the marks of syphilitic infection, such as bullæ and affections of the internal organs. Maceration alone is not indicative of syphilis. If the infant is born living, the evidences of syphilis are sometimes very few and equivocal. After a few months the diagnosis will sometimes be difficult; the eruption will have disappeared, leaving only an anæmia of uncertain origin, with a few discolored areas about the nasolabial folds and around the temporal region. There is a suspicious dirty-looking seborrhœa of the supra-orbital region. A rebellious anal eczema or copper-colored intertrigo which resists treatment should arouse suspicion. Pustular papules are not pathognomonic even if combined with joint-affections. A case came under my notice in which an infant had a varicella-like eruption with a painful swelling of the right elbow-joint. A diagnosis of epiphysitis syphilitica had been made and the eruption had been mistaken

FIG. 83.



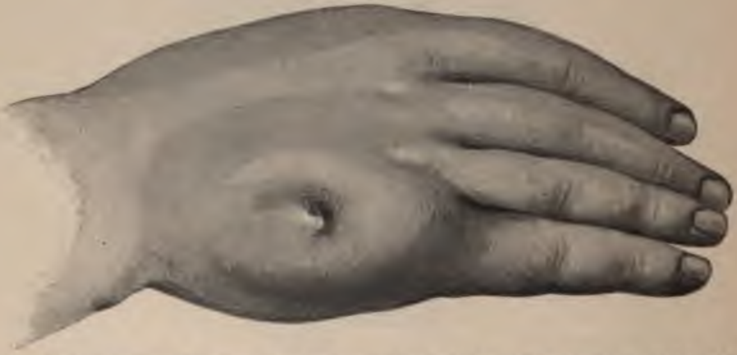
Hereditary syphilis: gummata of the cranial bones. Child, eighteen months of age.

for a syphilide. The color of the eruption was not that of a syphilide. Expectant treatment and immobility of the joint proved, after a few days, that the case was one of varicella with the joint-complication sometimes seen in that disease.

In the diagnosis of late hereditary syphilis the symptomatology is of service. In cases with bone-lesions it is often very difficult to differentiate it from tuberculous affections (Fig. 84). An active course of treatment then becomes necessary, with a view to diagnosis. This is especially the case in arthropathies, and also in late forms of dactylitis.

The **prognosis** as to life depends upon several factors. A breast-fed infant is more likely to survive than a bottle-fed infant. The possibility of complete restoration to the normal is slight. The majority of infants bear the marks of the disease into adult life, even under very favorable conditions of treatment and environment, and develop late in life the so-called late symptoms of hereditary syphilis. Some infants while progressing favorably under treatment, die suddenly without apparent cause; others remain stunted and delicate throughout childhood. Rachitis and its sequelæ seem to be very prevalent among infants who are the subjects of hereditary syphilis.

FIG. 84.



Tuberculous affection of the bones of the hand simulating syphilitic disease. Child, sixteen months of age.

The **treatment** of congenital syphilis may be either internal, by inunctions or subcutaneous injections. I have found internal treatment to be the most satisfactory. The effects of mercury are not so injurious as is the case with the inunction methods. The drug employed was calomel in combination with the saccharated ferric carbonate (this was a favorite remedy of Widerhofer):

Calomel	gr. ⅙ (0.01).
Ferri carb. sacc.	gr. iij (0.18).
Ft. pulver.	

A powder of this size may be given every three hours or four times a day. Some authors (Baginsky) prefer the protoiodide of mercury, grain $\frac{1}{6}$ to $\frac{1}{2}$ (0.01 to 0.03). If there is intolerance to calomel, satisfactory results may be obtained by the use of Lustgarten's preparation of hydrarg. oxydulatum tannicum, in doses of grains ij to v (0.1 to 0.3), repeated every three hours or four times daily.

If the rhagades, especially those about the anus, bleed or heal slowly, they should be stimulated with a weak solution of silver nitrate. Calomel should be dusted upon condylomata lata three times daily.

Baths of sublimate are recommended in severe cases of pemphigus, but it is not often necessary to resort to them.

Infants in the nursing period do not bear inunctions well. I have seen several cases treated by this method which lost weight rapidly or died suddenly, and this has been the experience of others (Monti). The old method was to place grains viij to xv (0.5 to 1.0) of unguentum hydrarg. under the flannel abdominal binder daily, and allow it to be absorbed, or the same quantity of ointment was rubbed in daily on various parts of the body.

Severe rhinitis is best treated by washing out the nasal passages once a day with a solution of corrosive sublimate (1 : 2000). The small glass syringe with a blunt soft-rubber nozzle is best for this purpose. After the syringing, unguentum iodoform. is applied to the interior of the nose by means of a camel's hair pencil.

How long should treatment be continued? No matter what method of treatment is adopted, mercury should be administered until all discoloration of the skin has disappeared. To attain this result will take a varying length of time in different cases. After the skin is clear and the anæmia has disappeared, it is well to cease the administration of drugs and observe the patient for further symptoms. Sometimes a patient will be brought to the physician for the treatment of a rebellious intertrigo long after all signs of general syphilis have disappeared. Such an intertrigo may have a copper color, and may ulcerate, the ulcers having a peculiar lardaceous appearance. In these cases, even if all other signs of congenital syphilis are absent, the internal administration of mercury gives brilliant results.

The treatment of late hereditary syphilis will depend much upon the nature of the therapeutic measures adopted earlier in life. In the majority of cases, the subjects being in later childhood or adolescence, it is well to begin treatment by a full inunction course, conducted on the same plan as with adult subjects with acquired syphilis. In addition, if gummatous affections of the bones are present, and if as in one of my cases visceral lesions, such as enlargement of the liver, have appeared, the patient is put upon gradually increasing doses of iodide of potassium. In one of my cases large doses of iodide of potassium failed to relieve the intense headache. This patient married, and after having a miscarriage gave birth under specific treatment to a healthy infant. The treatment of acquired syphilis does not differ from that of congenital or late hereditary syphilis.

XIX. ACUTE ARTICULAR RHEUMATISM.

(*Polyarthritis Rheumatica; Rheumatic Fever.*)

Although acute articular rheumatism is still regarded by some authors as a constitutional disease caused by disturbances of nutrition which result in local manifestations, the general tendency is to regard it as an acute infectious disease. The infectious agent, whether bacterial or toxic, attacks the serous cavities, such as those of the joints, the pericardium and endocardium, and the pleura. The resemblance of rheumatism, especially in children, to the infections is sufficiently great to warrant a serious consideration of this theory. Thus in septic endocarditis in children, as in the adult, there are symptoms of pain in the joints. Chronic cases of endocarditis of a rheumatic nature in course of relapse occasionally take a septic course. Certain diseases, such as erythema nodosum and peliosis rheumatica, in which the joint-symptoms are marked, are regarded as being caused by infection of a bacterial nature. I have lately seen such a case of peliosis. In other diseases, such as scarlet fever, measles, and varicella, there are joint-affections which are recognized to be of an infectious nature. Lastly, both American (Packard) and English writers have called attention to the well-observed clinical fact that there are forms of rheumatism and endocarditis which follow attacks of tonsillitis of the lacunar type or accompany them. It is true that the infectious agent, whether bacterial or toxic (Chvostek), is still to be discovered. Time may show that not one, but a variety of micro-organisms are capable of causing rheumatism of the acute articular type in a susceptible organism. Streptococci have been found in the exudate of the joints (Hlava). Staphylococci aureus, citreus, and alba have been found in the blood (Gutmann, Tizzoni, Bouchard). The pneumococci of Fränkel and the Diplococcus tenuis have been found in the joints (Leyden). Singer has found similar micro-organisms in the urine.

Heredity is among the predisposing causes. Children whose parents are markedly rheumatic, may suffer severely from the affection. Cold and exposure certainly predispose to the disease or precipitate attacks. The disease is common in countries such as England and America, in which climatic influences are favorable to its development, and is especially prevalent in the moist and cold seasons of the year.

Age.—Rheumatism has been described as occurring in early infancy (Jacobi). I have published a case in an infant of nine months. Rauchfus, Chapin, and others have also described cases in infants. These cases were collected by Miller, who, with his own case (nine months), found in the literature only 19 authentic cases in nursing infants. Although rare in infancy, rheumatism is not uncommon in children from the fifth to the tenth year. The majority of the cases of rheumatism occur between the tenth and the twentieth year.

Sex.—Among adults, males are more subject to the disease. In children, however, although certain observers contend that it is more prevalent among girls, other statistics show that it has the same frequency of occurrence in the sexes.

Symptoms.—Certain peculiarities, pointed out by Jacobi, seem to differentiate acute articular rheumatism of infants and children from the same affection in adults. But few joints are attacked. The pain and swelling are generally not very marked. The redness of the joint is slight or altogether absent. The temperature is rarely high. The smaller joints, such as the maxilla, sternoclavicular articulation, and those of the vertebræ, are rarely attacked. The larger ones, such as the ankle-, knee-, and wrist-joints, are most commonly affected.

Cardiac complication is the rule. As Jacobi has pointed out, endocarditis is sometimes the first manifestation of the disease.

Clinical Types.—In infants and young children the first signs are swelling and pain in the affected joints. The infant in the nursing period cries, has fever, and is restless. On investigation it is found that the patient favors one extremity, and shrieks with pain when it is touched. Children of two and one-half years or more refuse to walk, and will complain of the affected joint, ankle, or knee. There will be fever and constitutional symptoms. The ankle, and in some cases the smaller joints of the foot are swollen. One of the knees, the wrist, and elbow may also be swollen, red, and painful. The fever rarely rises above 103° or 103.5° F. (39.4° C.). In other cases there are fever and restlessness, and sometimes pains of an indefinite character in the joints. A history of pain may be elicited by careful questioning and examination.

The physician may find an angina, slight or marked; the heart may show signs of endocarditis of an acute type. There are pains in the joints but no true rheumatic swellings. The pains more closely resemble those in uncomplicated angina tonsillaris. In older children, a history of joint-pains with endocarditis may be obtained. In other cases, the pains in various joints are the only symptoms. There is no swelling or redness, and no endocarditis. Some cases have no fever. The classical cases, however, closely resemble those of the affection as seen in the adult. There may be premonitory symptoms, but as a rule the patient is brought to the physician with the enlargement of the joints fully developed. After the joints have become enlarged they may return to the normal in a few days, but may again be the seat of pain and swelling. The swelling in the joints of children does not persist as long as in the adult subject, and as a rule children are less disabled. In many cases there are gastric pains. The children do not show any greater tendency to perspire than adults.

Endocarditis is usually a complication of rheumatism in children. Its absence is rare. Only 2 of 15 of my hospital cases during the past year were free from cardiac complication. The most common cardiac lesion is found at the mitral valve and is manifested by a single systolic murmur at the apex. Three of the cases showed the presence of a double mitral murmur. Endocarditis sometimes does not reveal its presence by any symptoms, and is only discovered on a careful examination. In many of the cases there is also a pericardial friction first heard at the apex or base of the heart. The pericardial friction is more common in children than is generally supposed. The pericarditis frequently remains in the dry friction stage, and does not advance to effusion. Pleuritis and bronchopneumonia are among the less common manifestations. The endocarditis sometimes occasions pain and distress. The presence of endocarditis as an acute affection in first attacks of rheumatism has been dilated upon in the section on Endocarditis.

Chorea.—The relationship of chorea and rheumatism has been discussed. I have seen a child of two and one-half years born of a rheumatic mother, develop first rheumatism and endocarditis, and, within a few days, marked chorea. On the other hand, in many cases of chorea, there is neither endocarditis nor a history of rheumatism in children or parents. The statistics of chorea in hospital service show a greater frequency (39 per cent.) of cardiac disease with or without a history of rheumatism than the ambulatory cases. This is explained by the fact that only the severer cases of chorea come to the hospital.

The **prognosis** of acute articular rheumatism in infancy is good as to life. On the other hand, it is a disease which is likely to recur and to be complicated by endocarditis. The latter fact should cause the physician to reserve any definite prognosis until the course of the disease has been carefully studied. The prognosis of rheumatic endocarditis can never be definitely made. All depends on the amount of damage done to the valves and the frequency of the recurring attacks.

The **treatment** of acute articular rheumatism in children is not essentially different from that followed in the adult. I use salicylic acid, bicarbonate of sodium, salicylate of sodium, aspirin, and oil of wintergreen.

The bowels should be kept open with an alkaline cathartic. The Carlsbad salt or Rochelle salt given daily is best adapted for this purpose. The patient is put on a milk diet; fruit juices are allowed. The patient is kept in bed. The affected joints, if painful, are either immobilized or wrapped in cotton. Some prefer to paint the joints with a solution of oil of wintergreen, and then wrap them in cotton. Salicylate of sodium is given internally in doses of grains ij to v (0.12 to 0.3) according to the age. Young children are given a dose every three hours. Older children are given doses of grains vij to x (0.5 to 0.6). The effect is watched. Salol or salophen may be given. The salicylates sometimes not only act as irritants to the stomach, but also have no appreciable effect on the course of the disease. Aspirin has in my hands been useful in cases in which the salicylates were ineffective. In some cases I give bicarbonate of sodium in increasing doses until the urine becomes alkaline. Endocarditis is treated on the principles laid down in the section on that disease. While under treatment the patient is given alkaline waters. During convalescence the various preparations of iron are of great value. The preparations of lithium are useful in cases in which there are indefinite pains in the joints. The carbonate is given in doses of grain j (0.06) three times daily. It is given in capsule to older children after meals. The method of treating rheumatic subjects by the occasional administration of salol or salicylates for months has been suggested. The salicylates upset the stomach, so that the alkalies alone are available. The patient is given grains v (0.3) of sodium bicarbonate twice daily every other day. Vichy water is used regularly. In some cases the *tablets* of vichy taken once or twice daily are of great value.

Other Forms of So-called Rheumatism.

(*Rheumatoid Affections.*)

There are forms of joint-affection which it is not yet advisable to class with true articular rheumatism, but which are constantly and incorrectly called rheumatic.

The **gonorrhœal** form of rheumatoid affection is seen in infants and children who suffer from gonorrhœal vulvo-vaginitis or urethritis (Hartley, Koplik, Moncorvo). It may be monarticular or many joints may be affected. It is not, as a rule, combined with endocarditis. I know of no such case in the literature.

Peliosis.—Cases of so-called peliosis rheumatica closely resemble acute articular rheumatism. I have seen several in older children. In one, there were for weeks repeated painful swellings of the joints, with purpuric eruption about them. The gastric pains and critical sweats so often seen in rheumatism were present. These cases rarely present a temperature above 100.5° F. (38° C.). They show no cardiac lesion.

Tonsillitis with Joint-pains and Endocarditis.—Under the proper heading I have referred to cases of tonsillitis with indefinite pains in the joints and complicated with endocarditis.

Erythema Nodosum.—I have seen many cases of erythema nodosum in children. In all, the typical painful swellings on the anterior aspect of the tibia were present. There were also joint-pains, but in only 5 cases could I establish the presence of an endocardial murmur. I am therefore not willing to accept without reserve the contention of French authors that endocarditis is frequent in these cases.

The so-called **subcutaneous rheumatic nodules** are seen in children less frequently in this country than in England. They occur in endocarditis, and were present in 20 per cent. of Coult's cases (Donkin). They may be present in the absence of fever or in the febrile stage of rheumatism. They may be minute or of the size of an almond. They appear in crops, and may alternately appear and disappear for weeks. The nodules occur about the joints, elbows, knees, patella, over the vertebræ and scapula, and are freely movable under the skin which is not discolored. I have seen them in a case of rheumatoid arthritis, and also in one of peliosis rheumatica.

Muscular rheumatism is rare in infancy and childhood. Henoch describes cases of contracture of the muscles of the neck and of the nape of the neck. Among such contractures are forms of torticollis which are said to have a rheumatic origin. I have met many cases of torticollis in which with the contracture there was swelling of the cervical lymph-nodes. In such cases I have found eczematous affections of the scalp. It is possible that there was an acute infectious neuritis or myositis. There may, however, be cases resting on a purely rheumatic basis. All forms of torticollis due to hæmatoma of the sternomastoid muscles or to cervical bone disease, glandular disease, or neuritis should be excluded before a definite conclusion is reached. Henoch also refers to contractures of the abductors of the thigh which are of rheumatic origin. I have never seen cases of the kind.

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SECTION V.

DISEASES OF THE DIGESTIVE SYSTEM.

I. DISEASES OF THE MOUTH.

Physiological Facts.—The mouth of the infant up to about the eighth month is devoid of teeth, and thus nature indicates that the infant is not prepared to masticate solid food. The salivary glands show very little activity in the first three months of infancy, the secretion of saliva at this time being small in quantity.

In the newborn, before it has partaken of food, the reaction of the secretions of the mouth is neutral or slightly alkaline. Though an amylolytic ferment is present in the secretion of the parotid gland in the first days after birth (Zweifel), the function of this ferment is as yet a matter of speculation, inasmuch as the food of the newborn breast-fed infant contains nothing in which the action of such a ferment might be manifest.

Of interest is the act of nursing, which in the infant takes the place of the process of mastication.

Physiology of the Act of Nursing.—If an attempt is made to feed the newborn infant with fluids either from the spoon or pipette, there follows an abortive attempt at swallowing, accompanied by choking; it thus requires some skill and practice to induce the newborn infant to swallow fluids administered in this way. Not so with the breast. The newborn child instinctively takes the nipple of the breast, and nurses without the slightest education or preparation. The act of nursing, therefore, is purely reflex.

Thompson has described the so-called lip reflex: If the infant at rest or sleeping is gently tapped or touched on the upper or lower lip in the neighborhood of the commissure, there follows first a reflex movement on the part of the lips. If they have been separated, they close and form themselves into a pouting position; in other words, they purse themselves as if in readiness to take something into the mouth. The breast-nipple, therefore, performs a function for the infant similar to that of the finger in producing this so-called reflex of the lip. The nipple once having touched the lips of the infant is received by the pursed lips into a funnel-shaped opening, and the lips grasp the nipple and some of the adjacent skin.

It is received between the hard palate above and the superior surface of the tongue below. The lower jaw aids in making the contact between the lips and the nipple complete. The act of nursing itself, therefore, is the establishment, first, of a negative pressure, caused by the act of suction, equal to 0.5 to 0.9 centimetres of mercury. This alone would not determine the flow of milk into the mouth of the nursling were it not for the muscular pressure from below of the lower jaw. The combined force of the negative pressure produced by the act of suction and the muscular pressure from below on the nipple as it joins the breast is equal to 4 centimetres of mercury. This has been shown experimentally to be quite sufficient to determine an equable flow of milk from the breast into the mouth of the nursling. It takes from three to four acts of suction and muscular pressure to fill the mouth sufficiently to cause one act of swallowing on the part of the infant.

Characteristics of the Normal Mouth.—There are certain localities of the mucous membrane of the mouth which are especially liable to aphthæ or ulceration. Among these we must mention the mucous membrane over the hamular process of the palate bone, where it is normally paler than the surrounding tissue. This pale area on either side of the median line may be the seat of the so-called Bednar's aphthæ. Midway in the raphé of the hard palate in most newborn infants are seen one or two, at most three, yellowish-white, sago-like objects; these are called Epstein's pearls, because they were first described by this clinician. They are collections of epithelial cells, the remains of embryonal formations. These epithelial pearls are quite susceptible to traumatism, and if injured in any way become the seat of ulceration. Laterally on the hard palate over the alveolar process, above and below the mucous membrane is thin and has a white reflex. Any slight traumatism in this locality may cause ulceration. The tonsils of the newborn infant are scarcely visible. The posterior pharyngeal wall is glossy, of a bluish-pink color. On closer examination of the fauces of infants, bodies resembling drops of dew or vesicles are seen just in front of the tonsil. These are collections of lymphoid tissue, and are normal to the infant's mouth. They may become inflamed and form aphthous ulcerations, and when so inflamed are called herpes of the tonsil. There are also visible on the soft palate of children minute miliary, transparent bodies resembling vesicles, which are likely to enlarge in any disease affecting the mucous membrane of the mouth, as in the exanthemata. These also are aggregations of lymphoid tissue.

The bacterial flora of the mouth of the infant have been the subject of investigation by Lewkowicz, and include several, the streptococcic, micrococcic, and bacillary. Only the leading flora can be mentioned here: the pneumococcus, which is constantly present but not pathogenic; the streptococcus, in long chains similar to the pyogenic variety but not pathogenic; the Streptococcus salivæ of Veillon, the Streptococcus aggregatus of Seitz, the Staphylococcus pyogenes albus, the Streptococcus intestinalis or enteritidis of Escherich, the Micrococcus candidans (Flügge), the Bacillus acidiphilus of Moro, the most constant and frequent of the bacillary group; and the pseudodiphtheria bacillus. There are also, strange to say, anaërobic bacteria to be found in the mouth of infants, the most important being the Bacillus bifidus communis of Tissier. In all there are 23 varieties of bacteria normal to the buccal cavity of nursing infants.

Normal Dentition.

The teeth, both milk and permanent, are contained in the so-called tooth-sacs, which are situated in the alveolar process of the upper and the body of the lower jaw. The formation of these sacs begins in the sixth month of fœtal life, by a coalescence of the folds and papillæ formed in the jaw. There are twenty milk teeth, and the sacs of the permanent teeth are situated against the posterior wall of the sacs of the milk teeth, and probably communicate with them. As a result of the growth of the roots of the teeth, the milk teeth are pushed through the cartilaginous border of the jaw and the mucous membrane, and thus appear externally.

The eruption of the temporary or milk teeth begins about the sixth or seventh month with the lower incisors, and ends about the third year with the posterior molars. The eruption of the teeth, even in normal infants, varies within wide limits, some infants being precocious and others late in this process, without necessarily showing any signs of bone disease, such as rachitis. We might group the eruption of the milk teeth into five groups, as follows: The first group would include the two lower incisors, which erupt at from the seventh to the ninth month. There is then an interval of from three to nine weeks, when the second group, consisting of the four upper incisors, appears from the eighth to the tenth month. After this there is an interval of from six to twelve weeks, when the third group appears. This consists of the first molars and two lower lateral incisors, which erupt from the twelfth to the fifteenth month. An interval of three months then occurs, and the canines appear in the fourth group from the eighteenth to the twenty-fourth month. There is an interval of two months, and the four second molars appear. At the fifth or sixth year the third molar appears, and then the second dentition begins.

As exceptions to the above order, we may have the two upper lateral incisors delayed until the sixteenth month; the two upper incisors and the four posterior molars may be delayed as late as the thirty-sixth month. At the twelfth month an infant should have the four upper and two lower central incisors, with two lower lateral incisors coming. The lower incisors may not appear until the eighth or ninth month, and then be followed rapidly by others. I have seen several infants with one or two incisors at birth; they, as a rule, were imperfectly formed and resembled canines. These prematurely erupted teeth should be extracted if they interfere with nursing and lacerate the nipple of the breast. In some cases the upper incisors may appear first, and rarely canines may appear before molars.

The second dentition begins at the end of the sixth or seventh year with the eruption of the first molar behind the second temporary molar. The milk teeth at this time loosen because their arteries become obliterated, the nerves disappear, the alveolar sacs enlarge, and they fall out or may become carious. The permanent teeth appear in the second dentition, as has been said, very much in the order that the milk teeth appear—the central incisors about the eighth year, the lateral incisors at the ninth year, and the last molars from the eighteenth to the twentieth year, or even later.

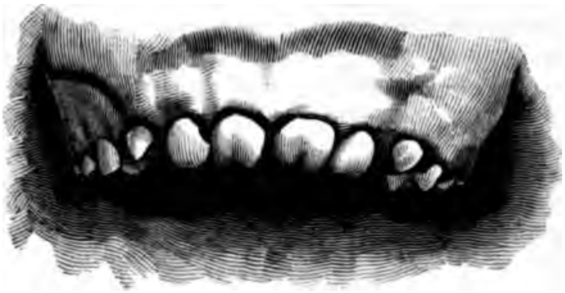
Abnormal Dentition.

Rachitis is a common cause of delayed dentition. Artificially fed infants are backward in cutting their first incisors. It is common to see bottle-fed infants cutting the lower anterior incisors at the ninth month. The infants may be in other respects normal. Rachitis affects the teeth of the first dentition mostly, but may influence the form and structure of the teeth of the second dentition. The teeth of the first dentition in rachitis are easily broken and are unnaturally white. In many cases the anterior incisors show an incurvation on the lower cutting edge, which is often mistaken by the inexperienced for Hutchinson's deformity. The first teeth in rachitis are easily eroded. It is not uncommon to see a rachitic infant with its whole dental system in process of decay. The permanent teeth present abnormalities in inordinate size and longitudinal furrows.

Syphilis.—The permanent teeth are affected by syphilis in a characteristic fashion.

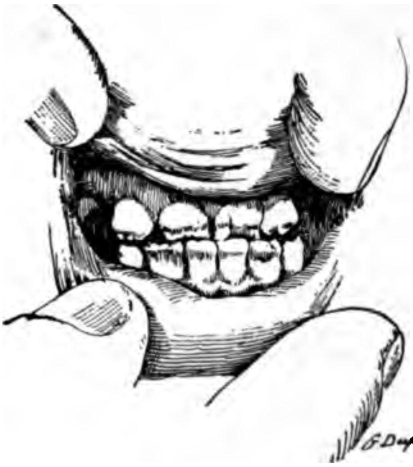
Hutchinson's teeth are so called because they were first described by Jonathan Hutchinson. They are the only teeth of the permanent set which are pathognomonic of congenital or very early acquired syphilis (infancy) (Fig. 85). In a large experience with syphilis in infancy and childhood I have seen but few perfect examples of these teeth. The teeth presenting the deformity are the central upper incisors of the permanent set, and these only. "These teeth show a central single, rather broad notch." In this notch the dentine, lightly covered by enamel, is exposed. It is seen as a ridge in the incurvation. The teeth are shorter and broader than is natural, and almost always have their angles sloped off. They are thus narrower at their cutting edge than higher up. They are seldom or never of good color, and frequently are not placed quite straight, but slope either toward or away from each other. Teeth which are the seat of erosion may resemble Hutchinson's teeth (Fig. 86). Fournier has described teeth in the temporary set which closely resembled Hutchinson's teeth. I have met an exquisite example of such teeth in an infant sixteen months old, the subject of syphilis (Fig. 87).

Fig. 85.



Hutchinson's teeth in a boy, twelve years of age.

Fig. 86.



Permanent teeth deformed through stomatitis in early childhood, resembling Hutchinson's teeth. Female child, nine years of age.

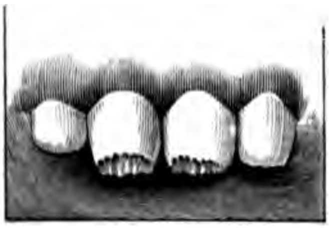
In syphilitic subjects we find the following deformities in the permanent teeth. These peculiarities are not characteristic of syphilis alone, but are found in those who are not syphilitic, but have suffered from stomatitis or dyscrasia of some kind. The changes are bilateral and symmetrical.

FIG. 87.

Central upper incisors of the first dentition resembling Hutchinson's teeth. Syphilis of the flat and long bones. Child, sixteen months of age.

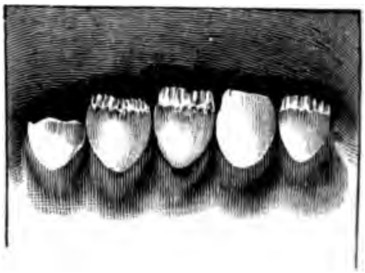
Dental Erosions.—The most important erosions, such as those of Hutchinson just described, affect the central incisors. Other erosions give the teeth an incurvated appearance on their cutting edge. In this incurvation is seen a supernumerary crown ribbed in a longitudinal direction (Figs. 88 and 89). The whole may be mistaken for Hutchinson's deformity. They result from malnutrition or stomatitis with faulty formation of dentine and enamel deposit in the eruptive period of the permanent teeth. The first molars show very characteristic deformities, which Fournier places next in importance to those of the Hutchinson teeth, but does not regard as pathognomonic of syphilis, although they are met in syphilitic subjects. This deformity of the first molars is shown in Fig. 90, taken from a child who showed other erosions, but gave no history of syphilis. I have seen these erosions very well marked in children who had positive syphilitic manifestations. The top of the crown is constricted, and there appears to be a double crown. Erosions are also seen in the canine teeth.

FIG. 88.



Upper central incisors, with erosions not syphilitic.

FIG. 89.



Lower incisors, with erosions not syphilitic.

Child, eight years of age.

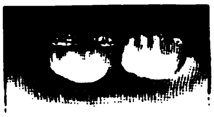
Microdontism.—The teeth are quite small, but if cared for remain perfect in shape, pearly and transparent. They are seen in children whose parents may have suffered from syphilis. The children may also have obstinate eczema of the anus (parasyphilitic). Microdontism may occur also as a result of any non-syphilitic dyscrasia.

Dental infantalism, described by Fournier, occurs in children who are syphilitic. Small teeth presenting erosions are interspersed among teeth which are normal in size and shape.

Amorphism, or the tendency of a tooth, such as the incisor, to take the shape of a canine, has been noted by Fournier. I have also met with cases of this deformity in congenitally syphilitic children. It is seen in children who have had syphilis, but may be met with in those who have no such history.

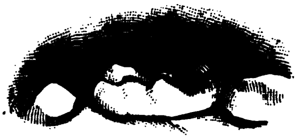
Children, subjects of syphilis, do not always present deformities of the teeth. In a girl of fourteen years, who gave a history of infantile syphilis, and who had late manifestations, such as gummata in almost all the bones, joint-affections, and gummata of the liver, the teeth, both upper and lower, were normal, of great beauty, and well preserved.

FIG. 90.



Erosion of molars, not necessarily syphilitic.

FIG. 91.



Molar tooth, showing erosion at crown. Boy, twelve years of age; same patient as with Hutchinson's teeth.

Pathology of Dentition.

The period of infantile dentition is one of great physiological activity and growth. The organism is forming at this time. The nervous system is in a condition of instability. The gut is exposed to all varieties of infection, and is very susceptible to them. During this period the infant or child suffers from a number of diseases and exhibits a variety of symptoms which in former times were difficult of interpretation. With advancing knowledge and the possibility of making more accurate diagnoses than were formerly feasible, the diseases incidental to dentition have become more a matter of speculation. There are clinicians of note who still believe that irritation of the trigeminal branches by an erupting tooth may cause reflex eclampsia. It is difficult, and not necessary, to pass here on the status of that section of infantile pathology which treats of the disorders incident to dentition. In the presence of mystifying symptoms the physician should make a very careful examination, in order to make the diagnosis. Clinical observation of a case for a few days, and accurate registration of the pulse, respiration, and temperature every three hours, may show that the diagnosis of dentition must give way to something more tangible.

Should the Gums be Incised?—I have often found the tooth-sacs to be swollen and the seat of painful distention just before the eruption of the teeth. In one case the tooth-sac was distended by a hemorrhage into its cavity. Under these conditions I have never yielded to the entreaties of the mother to lance the gums. I have seen no ill effects result from this *laissez faire* method. Very painful ulcerations result from friction, and uncontrollable hemorrhage may follow incision. In cases in which the sacs are distended, the functions of the stomach and gut should be kept normal, in order that complications may not be added to existing conditions. In rare cases I have seen suppuration in the tooth-sac, and have incised. In some cases of scurvy the tooth-sacs are distended and bluish in appearance. Treatment of the scurvy improves this condition.

Ulcerations or Erosions of the Angles of the Mouth.

(Fr., *Perlèche;* Ger., *Faule Ecken.*)

Definition.—This is a form of non-specific ulceration or rhagade occurring at the corners of the mouth, affecting the vermilion border of the mucous membrane.

Occurrence.—This infection is seen in children who present other signs of malnutrition, such as scrofulosis or lymphatism. They are anæmic, suffer from nasopharyngeal catarrh or skin eruptions, and live in unhygienic surroundings. The disease is seen in children under two years of age, and mostly beyond that period. The disease is confined to the corners of the mouth, and may be strictly limited to them, though the author has often seen it combined with erosions of the alæ nasi.

Symptoms.—These erosions, fissures, or rhagades consist of lineal ulcers of the corners of the mouth, which may have a red base and elevated borders, or the base and borders may have a bluish tinge, resembling mucous patches. In these children the question of diagnosis of these rhagades from those due to syphilis is constantly arising. The induration of the base of the ulcer which is present in syphilis is absent in the non-specific rhagade. The surface of the ulcer has a more lardaceous appearance in syphilis as a rule, the lips are involved, and there are mucous patches elsewhere.

The infection which we are describing is found isolated and limited to the corners of the mouth. The borders of the rhagade may be surrounded by minute pustules. The infection is symmetrical, involving both sides of the mouth. It is not painful unless the mouth is put on the stretch or acid substances applied to the base of the ulcer. In other cases the borders of the rhagades are raised and indurated. I have seen a large number of these rhagades; some, at least, so closely resembling a syphilitic lesion as always to warrant a careful exclusion in each case of this affection.

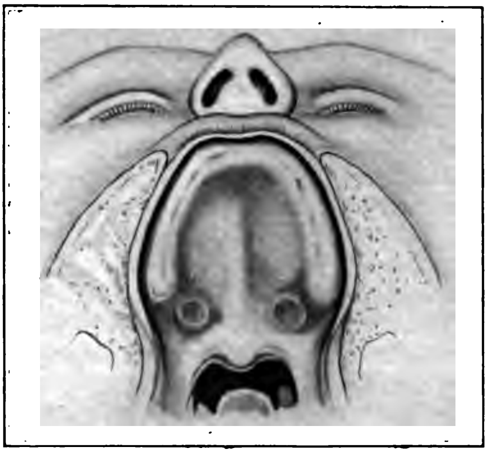
Diagnosis.—The diagnosis offers no difficulty, though it is an affection which rarely comes to the physician to be treated as an isolated disease, and is generally met in combination with other diseases. I have seen it in children suffering from typoid fever. The disease may be mistaken for diphtheritic infection, inasmuch as in some cases the base of the rhagade is covered by a pseudomembranous, whitish deposit. The culture tube will decide the true nature of the lesion in such cases.

Course.—The duration of the disease extends over a period of two or three weeks; if untreated, it usually becomes chronic. I have succeeded in curing these rhagades by touching them once daily with a 10 per cent. solution of nitrate of silver, and then applying the ointment of red oxide of mercury. Another remedy is the application of a solution of corrosive sublimate (1 : 2000).

Bednar's Aphthæ.

Bednar's aphthæ, named after the distinguished Viennese pediatrist who first described them, are two symmetrical ulcerations over the hamular process of the palate bone, seen in the newly born or very young infant (Fig. 92). In a large number of cases they are the result of traumatism. The finger of the nurse impinges on the processes of the palate bone when it is introduced into the mouth, and abrades the epithelium. Any bacteria which may be present in the mouth or on the finger thus gain foothold and ulceration results.

FIG. 92.



View of the hard and soft palate. Lateral ulcerations—so-called Bednar's aphthæ.

Epstein has shown that in the newly born infant such ulcers may be the starting-point of a general sepsis.

The infant may refuse to nurse, or if it does attempt to do so, the pain caused by the act of suckling causes it to desist. There may be intestinal disturbance, manifested by greenish stools and caused by infection of the gut by the bacterial flora of the ulcerations.

Treatment.—The ulcer should neither be washed nor traumatized. The rest of the mouth and tongue should be washed gently twice daily with a saturated aqueous solution of boric acid. The ulcers should be touched once or twice a day with a 10 per cent. solution of silver nitrate applied with a small piece of cotton on an applicator.

Sprue.

(*Thrush; Muguet* (Fr.); *Soor* (Ger.).)

Sprue is a parasitic growth on the mucous membrane of the buccal cavity of the newborn or very young infant. It may spread to the nose in cases of cleft palate; in other cases it may spread to the pharynx, larynx, œsophagus (Parrot), and even to the stomach (Parrot, Henoch, Northrup). The latter situation is not favorable to its growth. The parasite has been found in the movements of infants suffering from the disease.

Nature.—Sprue is one of the mould fungi. Its classification by various authors varies with the species examined. Older authors classed sprue with the oïdium as Oïdium albicans. Rees, Grawitz, and Kehrer classified it as a Mycoderma albicans, consisting of conidia and mycelia. Plaut classifies it as a common mould fungus (Monilia candida).

In the early stages it presents large or small irregular whitish masses. These may at first be very minute, covering only the summits of the papillæ of the tongue. On the buccal mucous membrane they may be as large as a pin's head or coalesce into masses resembling curdled milk. They may be seen on the roof of the mouth, on the soft palate, tonsils, and posterior pharyngeal wall. If the affection is progressive, the tongue and inner surface of the cheeks become coated with a white closely adherent pellicle. In neglected cases the sprue may be of a yellowish color if sarcinæ are present, or blackish or grayish in hue if other fungi have obtained lodgement. Considerable force is requried to dislodge the growth from the mucous membrane, and the operation will cause bleeding and considerable pain and traumatism.

Occurrence.—The organism is introduced into the mouth from without. It is present in the vaginal secretions of the mother, and has been found on the breast nipple. An abrasion of the mucous membrane must exist in order that the fungus may obtain lodgement. It is therefore found in infants whose mouths have been harshly washed with unclean fingers or into whose mouths unclean breast or bottle nipples have been introduced after harsh washing. The fungus having gained access to the cement-substance between the epithelial cells, proliferates into the deeper layers of epithelium, and may even invade the underlying connective tissue. Sprue carries with it any other bacterial flora which may be present in the mouth. A perfectly normal mucous membrane is not vulnerable to sprue. The sprue conidia and mycelia are found in the secretions of the mouth of the normal baby. Sprue is seen chiefly in infants whose health is below the average, who are inmates of institutions, or who have been in unhygienic surroundings.

Symptoms.—The local symptoms are due to the presence of the growth. In mild cases the patches are few in number and very minute. In neglected cases not only is the whole mouth the seat of the disease, but also evidences of infections of a pyogenic nature occur in the form of erosions of the buccal mucous membrane, yellowish plaque-like ulcerations and fissures which bleed easily. There is also dryness of the mucous membrane which has not been attacked or which has been freed from the fungus. Sprue, in fact, causes distinct reaction of the healthy mucous membrane in the vicinity of its invasion. Infants, even in the early stages, suffer from mild disturbances of the gastro-enteric tract, manifested by vomiting and greenish movements. In neglected cases marantic symptoms are also present. Older writers (Parrot) believed sprue to be a causal factor in athrepsia. It is simply regarded as a complication.

That pain is felt is evinced by the lack of desire to nurse the breast. A febrile movement occurs if the intestinal tract is involved.

Treatment.—**Prophylactic.**—Everything that is introduced into the mouth of the infant should be scrupulously clean. If the infant is breast-fed, the breast nipple should be cleansed before and after nursing with a pledget of cotton moistened with boric acid. The infant's mouth should not be cleansed after nursing. In cases in which the roof of the mouth has been carelessly cleansed there are not only the aphthæ of Bednar, but also sprue and other aphthæ in the median line as a result of traumatism to Epstein's pearls. If infants are fed articially, the nipple of the nursing-bottle should be boiled in soda solution once. If these precautions are carefully observed, and unclean fingers never introduced into the infant's mouth, sprue will rarely if ever occur. The normal epithelium and normal secretions are safeguards against the fungus.

Curative.—The growth should be removed by cleansing the mouth gently three times a day with a saturated solution of boric acid. The utmost gentleness should be used. Even in mild cases the removal of the sprue may extend over a number of days, because the parasite quickly reproduces itself. I use one piece of absorbent cotton for the roof of the mouth, another for the tongue, and another for the cheeks and lips. If it can be avoided, the mucous membrane should not be caused to bleed. If aphthæ exist, they should be touched lightly with a 2 per cent. solution of silver nitrate. The bowels should be opened by an initiative mild cathartic. Everything should be scrupulously clean. The severe cases, in which there is a septic condition due to extension of the sprue to the gastro-enteric tract, occur chiefly in foundling asylums. The infants die of septic infections. In private practice the prognosis is good if the case is seen early and correctly treated. Baginsky recommends potassium permanganate (1 : 150); others recommend corrosive sublimate (1 : 2000), but boric acid will be found to be equally satisfactory.

Henoch describes cases of sprue of the stomach. This is admittedly rare, and occurs in the form of slightly prominent plaques. Parrot describes sprue of the gastric mucous membrane as not infrequent.

Aphthous Stomatitis.

(*Stomatitis Aphthosa.*)

In this condition there are formed on the soft and the hard palate, the mucous membrane of the gums and tongue, and on the inner surface of the lips and cheeks, small round yellowish superficial ulcerations. These ulcerations, which vary in form and number, may coalesce and form irregular plaques. It is a question whether the ulcerations are the remains of vesicles which have burst, thus exposing an ulcerated base, or whether they are primarily ulcers. I am inclined to the former view, for in the so-called herpetic aphthæ of the tonsils the natural development of the aphthous ulcerations can be observed to advance from the vesicular to the ulcerative stage. This condition is very common in infancy and childhood, and according to Monti is most frequent between the first and the third year.

The **etiology** is still obscure. Some authors consider aphthous stomatitis an acute infection derived from the gut, possibly caused by toxins generated in contaminated milk (Forcheimer, Ritter, Kmeriem, Schamtyr). Others, basing their opinion on bacteriological studies, regard it as a purely local affection. The clinical course of the disease tends to support the former view. It has been compared by Forcheimer and others to the so-called foot-and-mouth disease of cattle.

The condition may occur idiopathically or may complicate intestinal infection, the exanthemata, bronchitis, tonsillitis, and pneumonia. Some authors believe that the affection may be communicated to others by the secretions of the mouth.

Bacteriology.—The forms of bacteria most commonly found in the ulcerations are the various streptococci and staphylococci (Judassohn). Bernabei has found the pneumobacillus of Friedländer. As these bacteria are present in the normal secretions of the mouth, it is doubtful whether they bear a causal relation to the condition.

Symptoms.—These aphthæ vary from the size of a pin's head to that of a split pea. They are invariably surrounded by an areola of inflamed mucous membrane. The outline of the ulceration may be round or irregular; as a rule the ulcerations are superficial. At the line of junction of the teeth and gums they may show a tendency to bleed if touched. There is considerable pain, with salivation, and in young infants also a distinct febrile condition and green diarrhœal movements. In other cases there may be an accompanying angina with swelling not only of the lymph-nodes at the angle of the jaw, but also of those underneath the jaw. In addition there are loss of appetite, and restlessness at night.

Course.—In well-nourished infants and children the tendency is to limitation of the aphthæ and spontaneous recovery within three or four days. In marantic or badly nourished children in unhygienic surroundings, the aphthæ are likely to spread, the ulcerations presenting the appearance of a mixed infection. Such cases are difficult to control. As a rule, however, the disease runs its course without leaving any lasting ill results.

The **treatment** of the cases in which the ulcerations or aphthæ remain discrete and in which mixed infection does not occur is begun with a saline cathartic, such as magnesia, or a dose of calomel. The mouth should not be washed. Careless attempts to cleanse the mouth are likely to cause the aphthæ to coalesce and spread, and also to cause intense pain. I administer a small dose of ferric chloride, made up with glycerin, every three hours. In most cases this will suffice. The use of potassium chlorate should be avoided with infants. If the edges of the gums adjacent to the teeth are affected, the teeth should be gently washed three times daily with a weak solution of tincture of myrrh or a saturated solution of boric acid. If the aphthæ coalesce, they should be touched once daily with a 2 per cent. solution of silver nitrate. With intractable young children, care should be taken in washing the mouth not to traumatize the unaffected mucous membrane.

Toxic Stomatitis.

I have seen a number of cases of stomatitis caused by irritant poisons, such as potash and ammonia. The children so affected had attempted to drink a solution of potash or ammonia from a bottle left within their reach.

The **symptoms** were purely local. The mucous membranes of the lips had a characteristic œdematous, swollen, and transparent appearance, the buccal mucous membrane and the tongue were pale and œdematous, and the papillæ were erect and transparent.

The **treatment** was expectant. A mixture containing bismuth subcarbonate seemed to give most relief. On subsidence of the œdema the mucous membrane presented a dry appearance. Sometimes small aphthous ulcerations appeared, which healed under applications of a 2 per cent. solution of silver nitrate.

In one case, five years of age, symptoms of œsophageal stricture were present three months after the ingestion of the irritant. Strictures of the œsophagus are more common after the ingestion of potash or lye solutions than after corrosion by ammonia.

Ulcerative Stomatitis.

(*Stomatitis ulcerosa; Stomacace;* Ger., *Mundfäule.*)

Ulcerative stomatitis is a disease of the mucous membrane of the mouth, gums, and tongue, which is characterized by ulceration with a fetid odor.

The **etiology** is still obscure. Frühwald and Bernheim found bacilli and spirochætæ (spirilla) in the ulcers. The fetid odor of the breath was reproduced in the cultures of Bernheim. The bacillus is lanceolate in form and resembles the diphtheria bacillus. These bacilli and spirilla are probably identical with those described in 1896 by Vincent as occurring in hospital gangrene.

The affection is most common between the fourth and the eighth year. The period of infancy seems to be exempt, in my opinion because of the absence of teeth. It occurs in children who have been neglected or who have lived in unhygienic surroundings, and is therefore very common in clinics and dispensaries. In the milder forms there is a line of yellowish ulceration along the margin of the gums at the point of contact with the teeth, and the adjacent mucous membrane is red and inflamed. When the gums are touched either in washing or in examination, bleeding occurs. There is a fetid odor of the breath, the tongue is coated; some children have pain and loss of appetite, and a slight febrile reaction. In the severer cases there are deep ulcerations along the margins of the gums, which bleed on the slightest provocation. Ulcers with a greenish-yellowish base are seen along the border of the tongue and beneath it. In these cases the lymph-nodes beneath the body of the jaw are enlarged and painful as a result of the infection. The salivation, pain, and local disturbance are considerable, and the fetor is marked. The buccal mucous membrane at the points of contact with the teeth may be deeply ulcerated, indurations of the tissues of the adjacent mucous membrane being also present. Small particles of necrotic tissue are seen to flow away in the saliva. So great is the pain that some children refuse to open the mouth or partake of food. I have seen the teeth become loose and necrosis of the alveolar process occur in places. Under the latter condition there is much swelling of the tissues above and beneath the jaw (lymph-nodes). The tonsils may also be the seat of ulceration of the same character as that occurring at the lateral margin of the tongue.

Treatment.—Cleanliness is the first step toward lessening the intensity of the inflammation. The mouth is washed every three hours with a solution of potassium chlorate, made by adding a teaspoonful of the saturated solution to a small glassful of water, or with a 0.5 per cent. solution of formalin. Internally, liberal doses of ferric chloride, made up with glycerin and water, have given the best results. If there are extensive ulcerative processes along the gums, the line of ulceration is gently touched once a day with a 10 per cent. solution of silver nitrate. In addition, the patient must have an abundance of fresh air, and is given a nutritious fluid diet, with fresh fruits and a small allowance of wine.

Gonorrhœal Infection of the Mouth.

This affection, sometimes called gonorrhœal or blennorrhœal stomatitis, is an infection of the mucous membrane of the mouth by the gonococcus of Neisser. Infection occurs only in places where the mucous membrane has been injured. There may be an associated gonorrhœal infection of the eyes or the vulva and vagina. The infection may be introduced into the mouth by the fingers of the nurse or mother. If the mother is suffering from gonorrhœa, infection may occur at the time of birth or subsequent to parturition. The cases thus far reported (Rosinski, Kast) have developed from two to thirteen days after birth.

Symptoms.—The constitutional disturbance is slight; there is no fever, no pain, and no interference with suckling. The lesions occur on those parts of the hard palate most likely to suffer from traumatism and subsequent infection—the parts favored by Bednar's aphthæ, the median raphe in the alveolar processes of the hard palate, and the anterior two-thirds of the tongue. Inspection reveals yellowish-white patches, due to infiltration of the superficial epithelial layers of the mucous membrane with inflammatory products. There is no pseudomembranous formation, but a pultaceous thickening. There is little tendency to spread, and no inflammatory reaction of the adjacent mucous membrane. The discharge is so slight that the saliva remains clear.

Examination of the secretion from the patches on the hard palate (which are generally symmetrical) and on the tongue reveals the presence of abundant gonococci not only on the surface, but also invading the mucous membrane along the cement-substance between the epithelial cells. The infection differs from that seen in adults (Cutler), in whom great constitutional disturbance and severe inflammation of the whole mucous membrane of the mouth are combined with a profuse ichorous buccal discharge and with pain. The tendency is toward rapid recovery.

The **treatment** is limited to the enforcement of strict cleanliness, and to local applications of weak solutions of silver nitrate (2 per cent.). The mouth may be washed twice daily with a solution of protargol.

Pseudodiphtheritic Stomatitis.

This form of stomatitis was first accurately described by Epstein. It is seen in newborn infants who have sustained a traumatism of the mucous membrane of the mouth. An infection of the injured membrane with streptococci results in the formation of a membrane resembling that seen in true diphtheria. These cases occur in foundling-hospitals and amid unhygienic surroundings. The pseudomembrane is of a greenish-yellow hue, and may spread over the hard and the soft palate, the tongue, and the pharynx. It may involve secondarily the entrance to the larynx, as happened in the cases of Epstein, and the epiglottis and œsophagus as well. Gastrointestinal symptoms and secondary septic pneumonia are developed. The temperature may, as in other cases of sepsis, be normal, or even subnormal. As a rule, the lymph-nodes are not enlarged. The condition must be differentiated from sprue and aphthous stomatitis. Aphthous stomatitis does not show any pseudomembrane; microscopical examination will aid in differentiating this disease from sprue.

Treatment.—Inasmuch as these cases are of septic origin, their course is progressive. On the other hand, small patches of membrane may be limited by applications of a 10 per cent. solution of silver nitrate. The membrane should not be peeled off, nor should the mouth be washed out with the finger. Antistreptococcic serum is of no use in these cases.

Noma.

(*Cancrum Oris.*)

Noma is a specific bacterial affection which attacks the tissues of one or both sides of the face, resulting in gangrene and destruction of the soft and hard parts. Babes and Zambolovici differentiate it from all other forms of gangrenous stomatitis and gangrene, such as those described by Henoch as occurring on the vulva.

The **etiology** is still obscure. Investigations thus far tend to show that several conditions clinically similar have been found to have a diverse etiology. Babes and Zambolovici isolated a very minute bacillus, and by inoculation experiments in animals produced typical noma. They found that this bacillus extends through the mucous membrane of the mouth, especially that of the gums. Accompanying it are a large number of streptococci, spirochætæ, and other bacilli. The latter play an active secondary rôle in the production of the gangrene. Gangrene is caused by an overwhelming bacterial invasion of the tissues. The toxins produced cause death of cell-life and necrosis in mass. In another set of cases, Walsh found the bacillus of diphtheria. These cases would appear to correspond to those published by Freimuth and Petruschky, who found a bacillus identical with the diphtheria bacillus in cases of noma of the vulva.

The greater number of cases of noma occur after measles. It may follow any of the exanthemata, typhus, typhoid fever, or any disease through which the power of resistance to infection is lessened.

Symptoms.—Henoch and Baginsky hold that in many cases an ulcerative stomatitis has preceded the main affection. The disease begins on the mucous membrane and invades the cheeks from within. Henoch alone has seen it begin from without in the form of a phlegmon of the cheek. It is first seen as a small ulcer with a blackish-gray base on the buccal mucous membrane opposite the teeth, or it may begin as a vesicle with serosanguinolent contents. After a period of time varying from a few hours to three or seven days the tissues of the cheeks become brawny and œdematous, the œdema involving the eyelids and lips. A dark, livid area finally appears on the corresponding exterior surface of the cheek. This area becomes black and gangrenous. Perforation and spreading of the gangrene rapidly result. The jaw may necrose and the teeth fall out. The process may spread downward along the neck, involving the shoulder in an œdematous, emphysematous, gangrenous mass. The indurations of the tissues of the cheek occurring in many forms of stomatitis ulcerosa should not be confounded with this affection; in these forms of induration gangrene is absent. In all cases of noma a marked gangrenous odor pervades the atmosphere about the patient.

The general condition of many cases is astonishingly good at first. The children seem unconcerned, and sit up in bed and play. The organism finally succumbs to the toxæmia accompanying such extreme destruction of tissue. There may be a febrile movement (103° to 104° F., 39.4° to 40° C.). The swallowing of gangrenous products in some cases causes an intensely prostrating and uncontrollable diarrhœa of a septic character. There is little or no pain. Death results within two or three weeks, either through general toxæmia and heart failure or complicating pneumonia.

Occurrence and Prognosis.—From a study of the literature, noma is found to occur most frequently between the second and the seventh year. The mortality is very high—fully 75 per cent. (Woronichin).

Treatment.—The most diverse methods have been employed in an endeavor to arrest the progress of the affection. To support the strength of the patient is the first consideration; careful ventilation, antiseptic and deodorizing solutions to mask the gangrenous odor, good food, and wine, are all of service.

The local treatment varies. Some authors advise dusting iodoform on the gangrenous area; others advocate the use of caustic zinc pastes in order to determine the line of demarcation between the gangrenous and healthy tissues. The Paquelin cautery with knife-blade attachment has been employed to remove the gangrenous tissue. Solutions of boric acid, thymol, and salicylic acid, should be freely employed to keep the mouth and parts clean.

In those cases, probably a distinct set, in which the bacillus of diphtheria is found, the antitoxic diphtheria serum should be injected in proper doses.

II. DISEASES OF THE TONGUE.

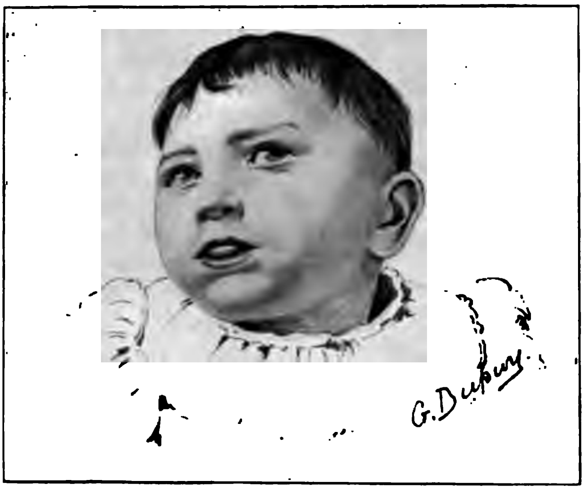
Congenital Anomalies of Size.

(*Macroglossia.*)

The tongue of some infants who are otherwise normal is unusually large and protrudes slightly from the mouth, but is of normal shape. It is pointed, but somewhat thickened in the middle (Fig. 93). As the infant grows older this anomaly becomes less apparent. In extreme cases the tongue protrudes from the mouth as a tumor mass. It is discolored—generally of a livid hue—and becomes ulcerated, especially at the line of the teeth. Infants thus affected cannot nurse, and the tongue must be reduced in size by surgical means. This congenital enlargement of the tongue may be due to an increase either in the connective or muscular tissues, or in both. In other cases the lymph-spaces of part or the whole of the organ are dilated—there is a lymphangioma of the tongue.

There are thus two forms of macroglossia—the one is called macroglossia lymphatica congenita, the other macroglossia congenita hypertrophica. The lymphatic form shows for the most part a gross hypertrophy of the organ and more rapid growth, combined with secondary changes in the lower jaw and teeth. The surface of the tongue is changed in appearance through defects of the epithelium and the results of inflammatory processes. The papillæ are enlarged, the organ is bluish red, nodular, not changed by muscular action and can be compressed. Speech is for the most part changed. The tongue in the hypertrophic form is smooth, the surface enlarged, the growth slow, the tongue less movable than normally and changed by muscular action. It cannot be compressed, as in the lymphatic form, and is less apt to become inflamed. The surgical procedures have consisted in compression, excision, and an ignipuncture, the latter being the most advisable (Eras). In cretins and the Mongolian forms of idiocy the tongue is also enlarged. It is broad, thick, and flat, and protrudes from between the lips.

FIG. 93.



Simple macroglossia.

Ringworm of the Tongue.

(*Wandering Rash of the Tongue; Lingua Geographica.*)

Ringworm of the tongue is a common affection of infants and children. It was probably first described by Santulus in 1854. Parrot regarded it as a symptom of hereditary syphilis—a view which has no clinical support.

In 103 cases reported by Böhm, the condition occurred sometimes in early infancy, sometimes as late as the twelfth year of life, and was most frequent between the first and the second year.

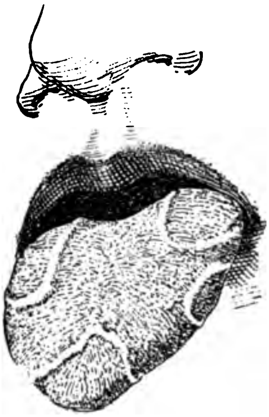
The **etiology** is obscure. Böhm believes it to be connected with a lymphatic diathesis (scrofulosis). It is found chiefly among children of the lower classes. It may, however, be seen in children in good hygienic surroundings and who are otherwise healthy.

If scrapings from the borders of the patches of an affected tongue be examined microscopically when fresh, large numbers of zoöglæa of coccus form, in some cases mingled with sarcinæ, will be seen. The presence of the latter micro-organism explains the yellow color of the border of the patches in some cases. The disease sometimes affects several children of a family.

The **symptoms** are limited to the appearance of the patches on the tongue. At the tip, but most frequently at the sides of the tongue, are seen areas sharply circumscribed by narrow, sinuous, perfectly oval or round borders (Fig. 94). The border is not only distinctly raised above the epithelium of the tongue, but also is of limited breadth and has a more pronounced whitish or yellow-white color than the rest of the tongue. Inside this border, if the patch is oval, the tongue seems to be denuded of its epithelium and is reddish in color. This condition should be differentiated from desquamation of the epithelium on the dorsum of the tongue, which presents a similar appearance, but in which the patches have not the band-like border (Fig. 95). Children do not appear to suffer inconvenience from this condition of the tongue.

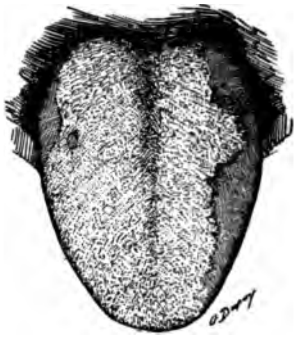
Treatment of the most diverse kinds, including local application of tincture of iodine and the use of ferric chloride, has in my experience failed to produce results.

FIG. 94.



Ringworm or wandering rash of the tongue, lingua geographica.

FIG. 95.



Epithelial desquamation of the tongue.

Desquamation of the Epithelium of the Tongue.

In this condition, which has been confounded with that just described, there are seen areas of irregular size and apparently denuded of epithelium. The boundary of these areas is sharply outlined, but the epithelium bounding the areas is apparently normal (Fig. 95). The tongue looks as if the epithelium had been scraped off. The condition demands no treatment, since it is only a symptom of mild derangement of the digestive processes.

Tongue-swallowing.

Tongue-swallowing is a term applied to a peculiar phenomenon seen in some infants who are the subjects of nasal obstruction. Infants normally breathe through the nose when at rest, the tongue being in contact with the roof of the mouth. If nasal breathing is obstructed either by swelling of the mucous membrane or by deformity of bone, the infant experiences great difficulty in breathing through the nose. As a result, not being accustomed to keeping the mouth open and the tongue on the floor of the mouth, the ineffectual efforts at nasal and mouth-breathing cause the infant to draw the tongue inward. The tip of the organ folds on itself, and may be drawn backward into the mouth in the efforts at mouth-breathing, causing a peculiar snapping noise to be heard on inspiration.

Treatment.—The remedy in these cases is nasal douching, and dilatation of the nasal passages with pledgets of cotton. The cotton is rolled around a probe or applicator, moistened with castor oil, introduced once a day into the nares, and allowed to remain about five minutes.

Tongue-tie.

Tongue-tie is a condition for the relief of which the physician is frequently consulted. Some mothers will ascribe inefficient nursing to this condition. With a breast secreting sufficient milk tongue-tie would not prevent nursing. The existence of the condition is readily detected if the organ is bifid at its tip when protruded. The frenulum will in such cases be seen to extend to the extreme tip of the tongue in a fan-shaped manner.

Treatment.—The frenulum being membranous is easily divided. It should be caught in the bifid groove of the pocket-case director and made tense, and the membranous portion divided with a pair of round-ended scissors. The ends of the scissors should be directed to the floor of the mouth. There is little bleeding. The infant should be placed at the breast directly after the operation, so that the act of suckling may stop the hemorrhage.

III. MALFORMATIONS OF THE UVULA.

The uvula is often bifid in infants. This condition is only of anatomical interest. There are cases in which the uvula is relaxed and elongated. In one case, in a boy five years of age, the uvula was so long that it gave rise to an incessant night-cough. On excision of the uvula the cough ceased.

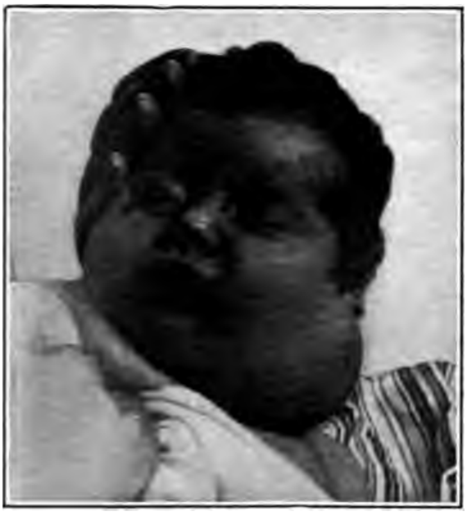
IV. DISEASES OF THE ŒSOPHAGUS.

Congenital Anomalies.

Branchial Fistulæ.—Among the congenital anomalies connected with the œsophagus is the so-called fistula colli congenita. This is due to a faulty closure of the branchial clefts in fœtal life. This fistula is generally unilateral, and is found at the inner side of the sternomastoid muscle. It may be bilateral. It generally leads to the pharynx or œsophagus, and may end in a blind canal. The canal may discharge mucus containing ciliated epithelium and leucocytes. Hennes described a cartilaginous growth in the neck, of which I have seen an instance. It occurs in the same situation as the above fistula, and is traceable to the same faulty closure of the branchial clefts.

Branchial cysts are cystic tumors of the neck and some parts of the head, originating from congenital defects of development. The primary origin of these tumors corresponds to the location of one of the branchial clefts, most frequently the second and third, in the vicinity of the larynx and pharynx. They are in intimate relation with the sheaths of the large vessels of the neck, the jugular vein, and carotid artery. The cysts are classified, according to their contents, into mucous, atheromatous, serous, and hematocysts. Branchial cysts are of rare occurrence. The *serous* variety is observed in

FIG 96.



Congenital branchial cyst. Infant seven months of age (Dr. Henry Heiman's case).

early life, either congenital or develops during infancy or childhood, whereas the *atheromatous cysts* are seen in early adult life. These cysts are seen most frequently on the left side of the neck. Their further consideration and treatment is of a surgical nature.

Diverticula of the Œsophagus.—These occur in childhood, are congenital in origin, and are accompanied by symptoms of difficult deglutition of solid foods, though fluids may be swallowed. In some cases the food collects in the diverticulum, causing swelling of the neck, with spells of coughing and consequent emptying of the diverticulum. With the difficulty of deglutition there is regurgitation of the food after eating. In a case recorded by Kurz there were undulatory movements at the side of the neck and gurgling noises heard on swallowing. A sound could be passed into the stomach, but at the junction of the upper third with the lower two-thirds of the œsophagus the sound passed into a pocket. In this case food could be caused to pass into the stomach while the patient was placed in a certain position.

The above diverticulum may be primary, of the congenital variety; or secondary, due either to a stricture of the œsophagus and dilatation above the stricture, or to traction from without on the œsophagus by a caseous lymph-node.

Congenital Stricture of the Œsophagus.—Sneider has collected 15 cases of congenital stricture of the œsophagus, most of which gave no symptoms during infancy and childhood. The stricture in these cases was either in the form of a ring of tissue or folds with thickening of the mucosa. They were present either in the upper or lower part of the œsophagus. Only 2 of the 15 cases died during childhood, the symptoms appearing for the most part in early youth.

The case recorded by Turner was that of a child eighteen months old. It had always suffered from difficulty in swallowing, and weighed only 14½ pounds. The mother said that since the period of weaning the child had become emaciated, and the difficulty in swallowing had increased so that finally all food was rejected. A sound having the diameter of the small finger could not be introduced into the stomach. Postmortem, the stenosis was found at the cardiac end of the stomach and was of the size of a No. 2 catheter.

Congenital Atresia or Absence of the Œsophagus.—The œsophagus may be entirely wanting, and in such cases other organs show anomalies; or there may be atresia of the middle third of the œsophagus; or the œsophagus may communicate in part with the larger bronchi. The stomach may be absent in some of these cases. In such cases the infants swallow, choke, have cyanotic attacks, and in three or four days cease to live. In one case published by Simon the œsophagus ran circularly around the trachea; the patient survived and died in adult life.

Œsophagitis.

Any inflammation of the mouth or the pharynx may extend into the œsophagus, such as croup, diphtheria, burns, corrosions, sprue. These affections cause no characteristic symptoms apart from the primary disease.

Caustic Œsophagitis.

(*Traumatic Stricture of the Œsophagus.*)

This is caused by the action of caustic alkalies or mineral acids on the tissues of the œsophagus, and the intensity of the corrosion varies with the amount and strength of the caustic taken internally. The caustic alkalies, such as potash and ammonia, are especially likely to be swallowed by children. The effects of the corroding agent are shown first externally. If a concentrated mineral acid has been taken, there is a brown or a black eschar. In less concentration we have white or grayish eschars, and later mild inflammatory reaction. Alkalies cause gelatinous swelling of the mucous membranes covering the lips, tongue, and buccal cavity. If the alkali be very strong, the tissues are converted into a yellow or brownish mass, and the fatal issue sets in before any reaction takes place. If the agent be dilute, superficial ulcers form after the primary corrosion. Reaction sets in, and, following the inflammatory stage of the reaction, cicatricial effects result, such as stricture.

The **symptoms** accompanying the swallowing of corrosive poisons are pain, which is constant, incessant crying, restlessness, due to a burning sensation in the mouth, attended with great pain and difficulty in swallowing. In some cases blood and purulent matter are vomited. There is great thirst. In other cases, where the concentration of the alkali has not been great, the lips are swollen, the mucous membrane of the mouth presents a whitish, gelatinous, swollen appearance. There is constant salivation; the children refuse to take solids or liquids, inasmuch as the least attempt at swallowing causes great pain.

The **treatment** of these cases is at first medical. Demulcents and milk are given in large quantities, and the physician should refrain from examinations with instruments lest perforation of the œsophagus or stomach result. After a few weeks, the primary effects of the corrosion having passed off and cicatrization of the ulcers having taken place, a stricture of the œsophagus results. The treatment of this stricture is surgical.

Periœsophageal Abscess.

(*Retro-œsophageal Abscess.*)

Griffith has reported 12 cases of this affection. It is not infrequent in infancy and childhood. The œsophagus begins above at the seventh cervical vertebra, lying in front of the spine. It passes behind the right bronchus between the two pleural sacs, behind the pericardium, and finally passes through the diaphragm. Any affection of the spine, pleura, pericardium, or lymph-nodes at the root of the lung may either cause pressure on the œsophagus, involve it in inflammation, or, if suppuration exists, the pus may break into the lumen of the œsophagus. Cases are recorded in which the pressure of an intubation tube or diphtheria of the pharynx has involved the periœsophageal tissue and caused abscess; or a foreign body in the œsophagus may cause perforation and ulcer, involving the adjacent connective tissue. If a foreign body is lodged in the œsophagus and is contaminated, as in the case of Soltmann, with actinomycosis, abscess of the œsophagus and lung may result, with actinomycosis of the latter organ. The most frequent cause, however, of peri- or retro-œsophageal abscess is disease of the vertebræ of a tuberculous nature.

Symptoms.—These will vary with the cause. An abscess of the pleura or a lymph-node pressing on the œsophagus will give symptoms of œsophageal stenosis. In some cases the pressure may interfere not only with deglutition but with respiration, and give rise to symptoms resembling laryngeal stenosis, necessitating intubation. As soon as the tube, however, is withdrawn from the larynx, the dyspnœa returns. The larynx may also be pushed to one side. There may be temperature, due to the primary disease. In one of my own cases there were spasmodic attacks of coughing, accompanied by cyanosis, and in one of the attacks a discharge of pus. The source of the pus in this case was probably an empyema which had opened into the œsophagus. These attacks were repeated at intervals, though with less expectoration of pus. The child finally made a good recovery.

In spondylitis there will be symptoms of disease of the vertebræ. If perforation occur from a bronchus or caseous gland, there are attacks of coughing, vomiting of food and pus, and finally symptoms resembling putrid bronchitis, and in some cases lung gangrene.

Diagnosis.—In some cases the diagnosis is not only difficult, but impossible. If the cause is evident and the abscess can be reached with the finger, the diagnosis can be made; but if the abscess is deep-seated, beyond the reach of exploring instruments, the disease is diagnosed only at the autopsy table. If the swallowing of a foreign body has preceded symptoms which resemble retro-œsophageal abscess, an x-ray should be taken to locate the body.

The **prognosis** in deep-seated retro-œsophageal abscess is bad; that in spondylitis likewise. The spontaneous rupture of the abscess, with discharge of pus externally and recovery, is exceptional. The spontaneous rupture of a retro-œsophageal abscess may result in pus finding its way into the larynx, thereby causing suffocation.

The **treatment** of retro-œsophageal abscess, if diagnosed promptly, is surgical. It may be stated, however, that these abscesses are best opened from without, and we should hesitate to make an internal incision in a deep-seated retro-œsophageal abscess.

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V. DISEASES OF THE STOMACH AND INTESTINES.

Classification.—The classification of the diseases of the gastro-enteric tract occurring in infancy and childhood must necessarily be schematic for the present, for much is yet to be learned, from chemical, physiological, and pathological standpoints, concerning some of these affections. Any classification, therefore, must be founded on a mixed etiological basis, and must, of necessity, be subject to future revision. For the present we may divide these diseases into:

First. Those which are caused by some congenital defect.

Second. Those which are purely functional, in which no anatomical lesion is supposed to be present, and in which, the cause being removed, the organ returns to its normal condition, no lesion remaining. Such are the acute dyspepsias of infancy and childhood, both stomach and intestinal; the various forms of vomiting, conditions of colic, and tympanites.

Third. A series of disturbances caused, it is supposed, not only by bacteria, but by their toxins, such as acute gastro-enteritis, summer diarrhœa, cholera infantum. In these diseases the anatomical lesion, if any exists, is in the majority of cases only temporary, for the patients recover. In the fatal cases the anatomical lesions are very slight and disproportionate to the severity of the disease, being due, it is at present supposed, to the direct action of the bacteria and their toxins on the superficial structures of the stomach and gut.

Fourth. Those diseases which are due to the direct action of the bacteria themselves, which, in addition to causing constitutional symptoms, due to the passage of the toxins into the circulation, also cause serious anatomical changes in the tissues of the gut, some of these changes causing eventually the death of the patient. In this class we would place dysentery of infancy and childhood, and the various forms of ileocolitis, which have, as yet, no firm etiological basis established by investigation and experiment.

Fifth. A series of diseases caused by some anatomical condition or neurosis. In this class must be placed the forms of congenital stenosis of the pylorus, dilatation of the stomach, which, though primarily caused by dyspeptic disturbances, eventually supervenes as the result of anatomical weakness of the muscular structures of the stomach. In this class we would place the various forms of constipation depending upon congenital dilatation of the colon.

THE STOMACH.

Anatomy.—The œsophagus enters the diaphragm at about the level of the ninth dorsal vertebra; the cardia is on a level with the tenth dorsal vertebra; the pylorus is in the majority of cases situated in the median line, but in some cases is slightly to the right of it. It is midway between the tip of the ziphoid cartilage and the umbilicus, and, being behind the liver, is not palpable. The stomach lies in an oblique position, passing from behind forward and downward. The pylorus is from two to two and one-half bodies of a vertebra lower than the cardia. In the newborn infant the inferior portion of the stomach has a fundus form (Pfaundler), which later becomes more marked. Occasionally there is no fundus, and the stomach is then of cylindrical shape. Between the time of birth and the seventh month the fundus of the stomach increases to fully twice its original length (Pfaundler).

The capacity of the stomach is still a matter of speculation. The absolute capacity, as given by Fleischman, Drewitz, Pfaundler, Holt, and Rotch, varies with the method employed to determine it. The work thus far done has been carried out on the cadaver, and, moreover, the methods employed presuppose an amount of pressure (14 c.c. to 30 c.c.) of water which does not exist in the normal state during life. The stomach contracts after death (systole); the distention with air or fluids is thus partly artificial. Lastly, the stomach capacity is of little aid in determining the point at issue —the quantity of food which should be taken by a healthy infant at each feeding. Figures giving absolute stomach capacity are useful only as indicating the actual size of the organ when full of fluid, a condition rarely present during life.

In the following table Pfaundler's results are compared with those of others. They were obtained by postmortem distention with fluid at a pressure of 30 c.c. of water. Fleischman distended the stomach at 14 c.c. of water pressure.

	Fleischman c.c.	Drewitz. c.c.	Pfaundler. cc.	Rotch. c.c.	Holt. c.c.
At birth	30	. .	30	30	36
One week	45				
One month	77	99	150	75	60
Two months	79	115	175	96	99
Three months	140	130	200	100	135
Four months	. .	165	230	107	150
Five months	290	253	260	108	170
Six months	260	297	295	. .	264
Seven months	. .	217	330		
Eight months	. .	289	365		
Nine months	. .	510	406		
Ten months	375	350	445		
Eleven months	. .	535	485	. .	243
Twelve months	. .	500	515		
One to two years	220	588	640		

Function and Motility.—The stomach of breast-fed infants empties itself in two hours after the ingestion of a full nursing. If the quantity of milk taken is small, a shorter time suffices. Bottle-fed infants taking cows' milk need fully three hours to accomplish the same result. This fact alone teaches that intervals of rest between the nursings, and a rest of four or five hours once in twenty-four hours, are necessary.

Marking out the Stomach by Percussion.—This procedure is difficult with infants and children. The normal stomach is rarely found outside of the left hypochondrium. The liver fully covers the stomach in the collapsed state. In the recumbent posture the stomach may be mapped out on the anterior abdominal parietes. It comes forward in the triangle formed on one side by the border of the left lobe of the liver and on the other by the border of the ribs. Above, the apex of the triangle is formed by a junction of the ribs and left lobe of the liver. Below, the base of the triangle is of variable length. In the axillary line the fundus in a moderately distended state is in contact with the thoracic walls, between the liver above and the spleen below. Above, it is separated from the lung resonance by a strip of dulness (the left lobe of the liver) which changes position with the movements of the diaphragm. The tympanitic resonance reaches downward in a vertical direction from the sixth to the eighth rib. Behind this, tympany is limited by the posterior axillary line; in front, by the triangle above referred to. I have frequently been able to confirm these statements of Fleischman. Anteriorly, I have with the aid of a gastrodiaphane shown that the transverse colon passes in front of the stomach just beneath the liver. It should be remembered that tympanitic resonance in the epigastrium is not always due to the stomach.

Acids of the Stomach.—When digestion is not in progress the stomach contains a tenacious, colorless mucus, neutral in reaction. When food is in the stomach, the reaction is acid.

Hydrochloric acid is normally present in the stomach of the infant (Leo, Van Putereu, Wohlman); lactic acid *only occasionally.* Heubner found 0.16 to 0.2 pro mille of lactic acid present. A considerable amount of hydrochloric acid unites with the salts and albumin of the milk, and is found as combined hydrochloric acid. When combination is no longer possible, the residue appears as free hydrochloric acid. The amount of free hydrochloric acid depends on the quantity of milk ingested, and varies from 0.8 to 2.1 pro mille. I have frequently failed to find it in the stomach contents of infants who are fed irregularly at frequent intervals. In healthy breast-fed infants free hydrochloric acid is found in from one and a quarter to two hours, and in bottle-fed infants in from two to two and a half hours *after nursing.* The effect of the lab-enzyme on the milk is marked in breast-fed as compared with that in bottle-fed infants. In the former the action of the acid delays that of the lab-ferment, while in the latter coagulation of the casein occurs in a short time and in large flocculi. The difference in retarding the action of the lab-ferment is due to the increased alkalescence of mother's milk, which requires more acid to neutralize the alkali, and thus to render coagulation possible: hence the greater digestibility of mother's milk.

Gastric contents containing free hydrochloric acid are antiseptic, while combined hydrochloric acid has no such properties.

Stomach digestion in the infant divides itself into three periods: The first, in which the milk is split by the lab-ferment into casein coagulum and soluble albumin; the second, in which the stomach contents become acid, having been previously neutral or alkaline, and in which chlorine combinations are entered into by the casein and lactic acid is formed; and third, in which the above phase of stomach digestion is completed, the contents pass into the gut and free hydrochloric acid appears.

Digestion is thus accomplished by a soluble ferment, so-called lab-ferment or pexin, which coagulates the casein of the milk; a soluble ferment, pepsin, which partly dissolves and peptonizes this coagulum; and chlorine combinations (HCl), which unite with the partially peptonized casein, and toward the end of digestion produce free hydrochloric acid. Thus the principal changes in the milk, so far as the stomach is concerned, occur in connection with the casein.

As soon as the milk enters the stomach, it is coagulated by the lab-ferment, whether its reaction is neutral, alkaline, or acid. This casein coagulation depends upon the lab and not upon the acid reaction of the stomach juice. Lab-ferment is present in the infant's stomach as such, and can be demonstrated in the stomach of premature and sick infants. Lab-coagulation of the casein is accomplished, according to Duclaux, in about fifteen minutes. Part of the casein coagulum is acted on by the pepsin and chlorine combinations and is converted into absorbable peptones (casease or caseon), the remainder passes into the intestine, where digestion is completed by the pancreatic ferments.

The casein coagulum of cows' and of human-breast milk are essentially different, the former being a firm mass, containing in its meshes the fat of the milk; the latter being in fine flocculi with little of the fat of the milk, and easily acted on by the stomach juices. In the bottle-fed infant the stomach, half an hour after feeding, still contains large coagula, whereas at this time the breast-fed infant's stomach contents consist of an easily absorbable homogeneous mass. Liquefaction is the work of the pepsin, which is present in the stomach juices of the newborn infant, though throughout infancy its action is weak and only sufficient to act on the proteids of the milk. Thus, half an hour after feeding, albumoses and peptones are found in the stomach both of breast-fed and bottle-fed infants.

Milk sugar is split partly into lactic acid about fifteen minutes after feeding, and by the action of lactase (Marfan) into glycose and galactose. This view, however, is not accepted by all observers, lactic acid not being admitted as normal to the stomach. The salts of the milk which have not been precipitated are directly absorbed. The fats enter, with the casein coagula, into the gut almost entirely unchanged, or a fractional part is saponified by lipase (Marfan) and absorbed in the stomach.

In general, it may be stated that in breast-fed infants digestion is completed in one and one-half to two hours: in artificially fed infants taking boiled milk in two and one-half to three hours, and in four hours in those taking raw milk.

The *bacterial flora* of the infant stomach are as yet not fully investigated. So far as known the stomach may contain the Bacterium coli commune, the Bacterium lactis aërogenes, the Bacillus subtilis and the related species, Tyrothrix granulatus and Bacillus butyricus of Hueppe, the Bacillus pyocyaneus, the Bacterium lactis erythrogenes, the Bacillus megatherium, the Spirillum rugula, a leptothrix, Staphylococcus pyogenes, Sarcina ventriculi, oidium, hay bacillus, and mould fungi.

Intestinal Digestion.—The stomach content of the infant as it is passed into the intestine consists of unabsorbed water; proteids which are made up of casein coagula and in part of syntonin; albumoses and peptones in combination with chlorides and ammonia; the fatty acids, leucin, tyrosin; and finally gases, especially carbon dioxide. There are present also the unabsorbed portion of milk-sugar and a small quantity of lactic acid. The fats pass into the intestine for the most part, suspended in the watery elements of the milk or entrapped in the meshes of the casein coagula. The whole stomach content has, as it passes into the intestine, an acid reaction, more marked in the case of the artificially fed as compared with that of the breast-fed infant.

The intestinal secretions concerned in the digestion of the above stomach content are those of the pancreas, liver, and intestinal wall (follicles of Lieberkühn and Brunner's glands).

Pancreas.—This organ is developed at birth, has a weight of 32 grammes or 1 ounce, and is, therefore, compared with the body-weight, much larger than in the adult. Whereas in the infant the pancreas weighs $\frac{1}{100}$ of the body-weight, in the adult it is $\frac{1}{600}$.

Ferments.—In the adult pancreatic juice there are three ferments—trypsin, ptyalin, and a fat-emulsifying ferment, steapsin. The infant's pancreatic secretion reveals trypsin and steapsin at birth, and even in the fœtal state. These ferments are present in small, but for the infant's uses sufficient, amounts. There is still a difference of opinion as to whether ptyalin is present at all in the pancreatic juice of the newborn. According to Karowin, a saccharifying power can be detected in the pancreatic juice not earlier than the sixth month of infancy, whereas Moro has found traces of such a ferment in the pancreas at birth. The fact of its absence or presence in but small quantity at birth has been brought forward as an argument against the use of amylacea in the food of the artificially fed infant at this age.

Liver.—The formation of bile begins at the third month of fœtal life, and at birth both bile and glycogen are found to be formed by the liver. The bile, which in quantity is comparatively greater at birth than in the adult, contains cholesterin, fats, lecithin, mineral salts, excepting iron. It contains small quantities of taurocholic acid, and but little or no glycocholic acid. It is not strongly antifermentative at this time. It contains bilirubin and biliverdin, and in the young infant urea. Its function in digestion seems to be limited to aiding emulsification of the fats.

The **intestinal juices secreted** by the follicles of Lieberkühn and the glands of Brunner are alkaline in reaction, and in the fœtus and newborn the ferments, present in these juices in the adult, seem to be absent (Miura). The rôle played by these juices in digestion is still a subject for study.

The principal process taking place in intestinal digestion of the infant is the transformation of the casein of the milk by the trypsin of the pancreatic juice into peptone and hemipeptone. Part of the casein is rapidly changed into peptone by the pancreatic juice, whereas the other portion is acted upon at great length, and from hemipeptone changed into absorbable substances which, partly crystalline, are taken up by the mucous membrane of the gut and synthetically transformed into albumins.

In the breast-fed infant the casein flocculi, being very small, are easily digested and dissolved in the duodenum, and the contents of this portion of the gut are slightly acid. In bottle-fed infants the digestion and solution of the casein is less complete in the duodenum than in the breast-fed infant, and the reaction of the contents of this portion of the intestine is distinctly acid.

The **milk-sugar** is split in the gut into galactase and dextrose and thus absorbed. This is accomplished, according to Marfan, by the lactase of the intestinal juices.

The **fats** of the milk pass from the stomach into the duodenum but little changed. They are suspended in the watery element of the milk or entrapped in the meshes of the casein flocculi or coagula. The fats are partly emulsified and in part split up by the pancreatic juice into fatty acids and glycerin, and in these forms absorbed by the intestinal villi. The digestion and absorption of the fats, however, is incomplete in the intestine of the infant, and much of it is excreted in the fæces in the form of neutral fats and fatty acids.

In the healthy breast-fed infant most of the above digestive transformation is completed in the duodenum, and the products are absorbed in the upper part of the small intestine. This is especially true of the casein or proteids, of which only traces are found in the lower portion of the small intestine.

Intestinal Residue.—After the absorption of the nutritive portion of the intestinal mess, the contents of the intestine consist of biliary remains, amido-acids, various products of bacterial fermentation, acids, and soaps, which are in part taken up and transformed by the liver and in part excreted. In addition, there are neutral fats and fatty acids. The minute quantity of proteids which has escaped digestion and solution and has not been absorbed is transformed by the bacterial flora of the gut into the products of decomposition, and as such are found as indol, skatol, phenols, and ammonia in the fæces. These also are in part taken up by the liver and in part excreted. The processes of decomposition, which are quite limited in the breast-fed and marked in the artificially fed infant, reach their highest development in the colon.

Characteristics of the Stools of Normal Infants.

It may be stated that the movements of bottle-fed differ from those of breast-fed infants in that they are lighter in color and in the main more bulky. In the perfectly normal breast-fed infant the stools may at times vary in color and general consistence; thus we can scarcely speak of a uniformly normal movement. Gregor has accounted for this by assuming that the stool of the infant at the breast may vary because of the composition of the breast milk from day to day and at different hours of the day. Inasmuch as the percentage of fat in breast milk varies so widely, the appearance of the stool will vary likewise. Infants fed on cows' milk and carbohydrates will have movements resembling those of breast-fed infants. If a number of normal infants are observed, it will be seen that from time to time even the breast-fed infant will present movements the consistence of which is more or less watery, and which contain coarse white curds and particles without any disturbance of the functions of the gut. Moreover, if we could receive the movements of a normal child on gutta-percha paper, it would be seen that the amount of water contained in a normal movement is very much more evident than would appear from its ordinary putty-like consistence on the diaper (Czerny). Infants taking a malted food will present movements that are dry and broken up into crumbs, and which have a distinct odor of malt. The movement of breast-fed babies and those fed upon carbohydrates and fatty food are softer than those of babies fed upon cows' milk exclusively. The movements of infants fed on cows' milk exclusively are lighter in color than those of the breast-fed child. The movements of the breast-fed infant have a distinctly acid odor, while those of the bottle-fed infant have an odor of decomposition.

In general the fæces of infants may be said to contain digested absorbable substances, indigestible substances, digested products of digestion and decomposition, anatomical elements of the digestive organs of the stomach and gut, mucus elements, and bacteria. If the movements of the breast-fed infants are closely examined, they are found to contain small whitish curd particles, the milk granules of Uffelmann. These were at first thought to be composed of casein: it is now known that they are made up of fat-crystals, and zoöglœa of bacteria. In addition, there are found in the fæces of infants epithelial elements, bilirubin crystals, and cholesterin plates. Fat appears in the fæces of infants rarely as fat crystals, but generally as fatty acids, neutral fats, and soaps.

The movements of infants fed on a mixed diet contain free starch-granules, cellulose, and also cholesterin plates and bilirubin; the products of decomposition—indol, skatol, and phenol—are also found, according to the time which has elapsed since the voidance of the movements (Blauberg). Sugar is not found in the fæces of infants, or only in small quantities (Uffelmann and Blauberg). Michael has found that the gross weight of fæces in the newborn breast-fed infant was about 1.5 per cent of the gross amount of food ingested; while later in infancy the movements were 2.7 per cent.

of the amount of food ingested. Rubner and Heubner found that in bottle-fed infants the fæces were about 4.7 per cent. of the amount of food ingested. Michael found that the fæces in the first days of infant life contained about 72 per cent. of water, while in the ninth month of infancy they contained 85 per cent.

Reaction of the Stools.—The reaction of the stools of infants, both breast- and bottle-fed, has been the subject of much discussion, because of the difference of opinion among investigators as to what constitutes a normal movement in an infant. It may be stated, however, that the stool of the breast-fed infant is regularly acid in reaction and has an acid odor even after being passed for some time. The infant fed upon cows' milk has a stool which is alkaline in reaction, sometimes neutral, and, under certain conditions which no longer may be looked upon as absolutely normal, slightly acid. The stools of these infants have an odor more or less recalling that of stale cheese; in other words, an odor of decomposition.

The Daily Number of Movements.—The normal infant, whether on the breast or the bottle, will have one, two, or even three movements daily when in perfect health. In the breast-fed infants these movements may be small or large and even contain quite an amount of fluid and still be within the limits of health. In the bottle-fed infants, however, the stools are, as a rule, larger in bulk than those of the breast-fed infants, and contain less water. I have seen bottle-fed infants in perfect health who have had as many as four movements daily, all having normal characteristics. Infants may have six movements daily and still be in perfect health. If the consistence and color are within normal limits, the number simply indicates the amount of intestinal residue, and not disease.

Bacterial Flora of the Intestine.—Two or three days after birth the meconium changes its characteristics and assumes those of milk fæces. In the milk fæces of the infant nursed at the breast we find as predominant, first, a bacillus described by Tissier, which stains with Gram's stain, and which in the crude specimen seems to occupy most of the microscopic field. This is called the Bacillus bifidus communis. It is an anaërobe. In addition to this bacillus, we find next in numbers the so-called Bacillus acidophilus of Moro and Finkelstein. The latter also stains with the Gram stain. In addition to these two bacilli, which are found in greatest numbers in the fæces of the breast-fed infant, we have a few coli bacilli, and also some numbers of the Bacillus lactis aërogenes.

The fæces of the infant fed on cows' milk present a much more luxuriant flora of bacteria than those of the breast-fed infant. There are: (1) the Bacillus coli communis, (2) the Bacillus acidophilus in small numbers, (3) other Gram-staining bacilli, (4) the Micrococcus ovalis (Escherich and Tissier), (5) the enterococcus of Thiercelin, (6) a diplococcus staining with Gram, (7) streptococci and staphylococci, (8) Sarcina minuta, (9) the Bacillus lactis aerogenes. The Bacillus lactis aërogenes splits milk-sugar into lactic acid, carbonic acid, and water, and causes the intestinal contents to become acid. In the lower part of the gut we find the Bacillus coli communis, a micro-organism which may exist in the presence of any reaction, and which splits milk-sugar into lactic acid, carbonic acid, and water, and partly splits fat into fatty acids. It is the prevalent micro-organism in the stools, though with it we have a number of the Bacillus lactis aërogenes, a yellow fluorescent or fluidifying bacillus, three fluidifying cocci, a Micrococcus ovalis, a porcelain coccus, the tetrad coccus, the white and red hay bacillus, a capsule bacillus, the Monilia candida, all of which exist in varying quantities.

Acute Gastric Dyspepsia.

(*Indigestion.*)

Acute gastric dyspepsia may clinically be divided into two forms, that affecting infants, and that affecting older children. The period of infancy is one of frequent disturbances. Mental excitement on the part of the nurse may cause the milk to disagree with a breast-fed infant. The ingestion of an undue quantity of breast-milk, even if of good quality, may cause indigestion. Certain articles of food, notably asparagus, if partaken of by the mother, may cause gastric irritation. Nursing a breast in which the milk has caked will also cause indigestion.

Symptoms.—Vomiting is the first evidence of disturbance of the digestive processes in the infant. It occurs after feeding, and is at first not accompained by constitutional symptoms or diarrhœa. If the exciting cause continues, a slight febrile movement is noted, and also slight prostration. The infant is restless, but having vomited is relieved, and if permitted will again take the breast, the vomiting taking place after each nursing. The bowel movements then become disturbed. They may not only be green, but also frequent and in some cases fluid. There are in all cases colic and tympanites.

Acute gastric dyspepsia in older children may be caused by some article of diet which has disagreed with the patient. The symptoms are much the same as those seen later in life. It is important both with infants and children to determine whether the symptoms are due to improper food or whether proper food has for some reason disagreed. Bottle-fed infants are liable to indigestion if the milk contains any extraneous substances, not necessarily toxic ones.

A baby may have thrived for weeks on a certain food-mixture, when suddenly, without apparent cause, symptoms of gastric dyspepsia supervene. In such cases it will be found that the acidity of the milk was greater than usual, or that the fodder of the cows furnishing the milk has been changed.

Course.—If the food is suspended and proper treatment instituted, the symptoms subside and the infant recovers, but if the exciting cause is not removed, more serious disturbance of the stomach and gut will develop.

Treatment.—It is best both with breast-fed and bottle-fed infants to discontinue the giving of all food as soon as symptoms of indigestion appear. With the suspension of food the administration of a simple cathartic (castor oil) is all that is necessary. The infant is put for twelve hours on a solution of white of egg, and the breast pumped regularly every three hours to prevent caking. The breast may then cautiously be exhibited. Stomach washing should not be resorted to, and the breast should not be denied for too long a period. If, on resuming breast-feeding, symptoms reappear, an analysis of the milk should be made. Its composition may have changed and too much fat may be present. We should not be hasty in taking an infant from the breast and placing it on the bottle on account of a few symptoms of gastric dyspepsia. Proper regulation of the diet and the taking of proper exercise by the nurse will frequently cause the desired adjustment of the constituents of the milk and the disappearance of symptoms.

Habitual Vomiting of Infants.

Habitual vomiting of infants refers to the regurgitation of milk in the uncoagulated state shortly after nursing. It occurs in infants in apparently good health, and is not followed by loss of weight or disturbance in the functions of the gut. Some infants vomit curdled milk in the same manner. The cause of this form of vomiting has been variously explained. The simplest explanation is, that by slight pressure the food is forced into the œsophagus and thence reaches the mouth. It is a well-known fact that the stomach of the infant can be emptied by gentle abdominal pressure. Another explanation is that on deep inspiration the negative pressure caused by descent of the diaphragm forces a certain amount of fluid from the stomach, which is almost vertical in the infant, into the œsophagus and thence into the mouth. This form of vomiting requires no treatment. The general impression is that it can be stopped by regulating the amount of breast-feeding, but this belief is erroneous, as the vomiting persists after such precautions have been adopted. Fleischman thinks that the habit is hereditary in certain families.

Cyclic Vomiting.

(Periodic vomiting ; Recurrent vomiting.)

Definition.—Cyclic vomiting is a condition in which there appear at intervals more or less remote from each other attacks of vomiting, accompanied by marked prostration without rise of temperature, in which there is an absolute intolerance of the stomach for even fluid food. This condition has been described under various headings both in France, 1841, by Dr. Gruere; by Lombare, in 1861; in England by Gee, and in America by Rotch, Holt, Rachford, Edsall, Koplik, and others.

The **etiology** of this condition is obscure, and it is most probable that the theory of Rachford is correct—that the *symptom-complex* is one of gastro-intestinal lithæmia, due to an increased acidity of the fluids of the body as the result of disturbed metabolism. In one of these cases Herter has analyzed the amount of uric acid in the periods preceding, during, and following the attack. In such an analysis the gross amount of uric acid was greatest on the second day of the disease, and fell rapidly on the third day to near the normal. The normal relation of uric acid to urea in these patients was as 1 : 54. During the attack the relation of uric acid to urea, as a rule, was 1 : 85, and in the normal condition it fell to 1 : 42. Griffith considers the condition a species of toxæmia.

It seems to me, from a study of a number of my own cases, that the condition described by Rachford must obtain; in addition, however, these are crises in which the patients seem to suffer distinctly from attacks of intestinal intoxication, inasmuch as treatment directed toward placing the conditions in the gut on a normal basis seems to benefit them materially. In most of my own cases there has been a history of constipation extending over long periods of time, and an intolerance of milk as the main article of diet in other cases.

Other authors (Holt) have not found constipation to be a prominent factor in their cases, but rather that the ingestion of certain forms of foods, such as amylacea, are apt to precipitate an attack. In only one of my cases have I found that amylacea were badly borne, and the ingestion in this case of a cereal gruel seemed to precipitate an attack; constipation, however, existed in this case from infancy.

The **symptoms** in these cases are quite characteristic; the subjects of this form of disturbance may be well developed, but, as a rule, they are pale. In some of them the anæmia is quite marked, and the children have a pasty complexion. The attack is preceded by a period during which the child complains of slight pain in the stomach; in some cases this may be absent. The child awakens in the morning, feels tired, has no appetite for breakfast, and has pronounced pallor. Vomiting sets in; the food is first rejected and then vomiting persists; in some cases even blood with mucus is vomited from the stomach. In other cases the contents of the duodenum may appear in the vomitus in the form of biliary matter. The child finds most comfort in lying quietly on its back, refusing to take any food; even water is vomited. There is no temperature; there may be a slight increase of the pulse-rate, and it may have a bounding character, and the heart-impulse may be increased in force. There may be a complaint of epigastric pain. The prostration in some cases is extreme; the condition may last twenty-four hours to two or three days, until normal conditions are established. The vomiting may recur several times in twenty-four hours; it gradually diminishes in frequency and disappears. During this time there is no movement from the bowels, or there may be a constipated movement as the result of enemata, with the voidance of a large quantity of mucus. The following is a characteristic case:

Dorothy E., five years of age, fed in infancy on modified milk; has never suffered from any disease of greater severity than a grippal attack. She has been constipated since infancy, and this constipation has lately become more marked. After having been put on raw milk and cream, the constipation abated for a few weeks and then returned. The constipation was only relieved by the constant use of cathartics, and sometimes these were not effective. The child is a well-developed girl, thirty-five pounds in weight, with a body-length of 102 cm. (3 feet 4 inches); the abdomen is protuberant; there is no disease of the heart or lungs; the liver and spleen are normal in size. The urine does not contain albumin or casts. The child is anæmic, has a tired expression, and in her movements there is considerable mucus. Her vomiting attacks began when she was four years of age. These attacks last two or three days, during which the child rejects all food. The attacks begin very much in the manner just described. In one of these attacks the vomiting was so severe that there was an alarming hemorrhage from the stomach. The odor of the breath in the first day of the attack is "sweetish" (acetone).

Some of my cases during the attacks presented albumin and a few hyaline casts in the urine. These disappeared after subsidence of the attack. Acetone bodies may be present in the urine in increased quantity.

Diagnosis.—The practitioner should be exceedingly cautious when presented with a case of vomiting in a child from four to five years of age not to hastily conclude that it is one of cyclic vomiting before making a thorough examination, not only of the urine, but of the other viscera.

A case has recently come under my notice, observed for four years, in which a diagnosis was made of cyclic vomiting, but which is one distinctly of nephritis with recurrent attacks of uræmia.

Some authors, such as Rotch, have laid stress on the fact that these attacks may also simulate meningitis.

Course and Prognosis.—The prognosis in this condition, so far as life is concerned, is good. There are some cases recorded which have terminated fatally. The author has not met in his experience such an unfortunate occurrence. The course of the disease, if properly handled, ends in recovery in from twenty-four hours to three or four days.

The **treatment** of cyclic vomiting is divided into the treatment of the attack and the intervals between the attacks.

Treatment of the Attack.—The patient is put to bed, kept perfectly quiet, and little or no fluid is given by the stomach—certainly no solid food. The stomach is quieted with small doses of codeia. This is the only remedy which in my hands seems to have had any influence in controlling prolonged vomiting. Enemata consisting of saline solution are given twice daily. They should be high enemata, and at least a quart of water should be thrown into the rectum at each sitting. In the intervals between the enemata the child should be nourished by the rectum. Somatose solution—1 drachm of somatose to 8 ounces of cold water—is heated to a lukewarm temperature, and given by the rectum in quantities varying from 2 to 4 ounces every three hours. The patient is given small pieces of ice to swallow, in the case of older children. No other treatment is necessary until the attacks of vomiting subside of their own accord within twenty-four hours. It is surprising to see how comfortable these little patients will be if little or no fluid is taken by the mouth; in fact, some of them are intelligent enough to find this out for themselves and refuse all nourishment. On the second day of the disease, when the vomiting has subsided to a great extent, we may give the patient broths, fruit juices, diluted gruels; and on the third day we may gradually return, if the stomach is tolerant, to a semisolid diet, and finally to a full diet. As soon as the stomach is tolerant of fluids, and even at the height of the attack, small quantities of Vichy given by the stomach seem to be grateful to the patient. During this period also the alkaline treatment, which will be spoken of, may be inaugurated; and finally we may, toward the close of the attack, if this is possible, give a vigorous cathartic, such as cascara, or Rochelle salts.

In the intervals between the attacks these patients do best on the following treatment: The bowels should be kept in a normal condition; if the child is constipated a rectal enema should be given daily, and, if this is not effective, it should be supplemented by some catharthic, such as cascara, in order to facilitate a complete daily evacuation of the bowel. The diet in these children should be a mixed one. I have found that whereas some of these children will not tolerate cereals others will. The rule, however, is that we should reduce the quantity of milk, especially in the older children, to a minimum, and, if possible, place the patient on a diet in which milk enters but little. They should be placed, so far as medicinal agents are concerned, on the so-called alkaline treatment, which has been found to be most successful in these cases. For a child from three to five years of age I prescribe a powder composed of 2 to 3 grains of bicarbonate of soda and ¼ to ½ grain of carbonate of lithium. This powder is given three times daily after meals in a glass of Vichy Celestins. The children are bathed daily in a bath in which a handful of bicarbonate of soda and a handful of salt have been dissolved, and are rubbed down after the bath with a very dilute solution of alcohol in water and a rough towel. The muscles of the body are kneaded, if a masseuse is available. Sojourn in the open air as much as possible is advised, and sports which involve muscular exertion encouraged. Regularity at meals is inculcated, and these little ones are taught, if possible, to evacuate the bowel regularly. In some of these cases the coarser the diet, the more successful seems to be the treatment, for in the most aggravated cases that I have seen there has been a too "finicky" selection of a few articles of diet for these patients, and the little ones have been kept in some cases on milk, gruels, and fruits, to the exclusion of everything else, for months.

Other Forms of Vomiting.—There are other forms of vomiting which are of interest in this connection :

a. Some children vomit when irritated or after outbursts of temper, or may vomit at will if their food or anything in connection with their discipline does not meet their approval. Some of the little patients know intuitively that vomiting alarms the mother, consequently it will appear whenever any concession is to be obtained in the nursery.

b. Vomiting, especially after eating, may be caused by a severe attack of coughing. If vomiting occurs frequently under these conditions, whooping-cough should be suspected.

c. The vomiting of pyloric stenosis of the congenital type is characteristic. It is more in the nature of a regurgitation. When lying on the back the baby vomits at intervals, and in small quantities. After a nursing there is an interval, after which the infant vomits two or three times the amount of food taken at the recent nursing. This is explained by the fact that in this condition there is some little vomiting constantly going on, due to the increased peristalsis of the stomach. There is, however, a small quantity of food retained in the stomach. This residual quantity increases with each feeding, and is finally rejected in the manner just described.

d. The vomiting of appendicitis is also characteristic. The patient is seized suddenly with sharp abdominal pain and then begins to vomit. The vomiting may recur once or twice, and then cease. In neglected cases, in the final agonal stage, vomiting due to sepsis and toxæmia may be persistent.

e. Vomiting is the first symptom in intestinal obstruction. It may be followed by a very small movement, and then for a short time there is, as a rule, no action on the part of the bowels. The vomiting may not recur in the first twenty-four or forty-eight hours, except at long intervals, but the bloody movements recur frequently, and pain is also present. The vomiting returns when the intussusception is more marked, and late in the affection becomes fecal.

f. Vomiting occurs at the outset of the infectious diseases. Persistent vomiting extending over a period of months is often of nephritic origin.

g. The vomiting which accompanies meningitis occurs at the outset in that of the cerebrospinal type, and is quickly followed by cerebral symptoms. In tuberculous meningitis it occurs at the onset and after the appearance of a vague series of cerebral symptoms. It is rarely persistent after the initial attack. The subsidence of the vomiting and the sequence of cerebral symptoms and a febrile movement will easily distinguish this form of vomiting from others.

Tumor and abscess of the brain are accompanied by vomiting at intervals.

Colic.

Colic is not a disease, but a symptom of disturbed conditions of the gut. It is really a painful contraction of the muscle-fibre of portions of the gut-wall. In the simplest form the painful contractions are incited by actual distention of the lumen of the gut. The pain caused in colic is in the majority of cases not of the character which arises in certain other affections of the gut which are neurotic in nature, nor is it of the same nature as that seen in enteritis. Pain similar to that in colic may be caused by the administration of some such drug as lead, arsenic, etc.

Cause.—In the great majority of cases the affection is caused by some disturbance of the processes of assimilation in the gut. It is uncommon in infants in good condition, and its appearance in any case indicates the necessity of an investigation into the condition of the digestive processes in the stomach and intestine. The form of pain or colic accompanied by distention (tympanites) seen in newborn infants, and also at the height of pneumonia in older children, has an etiology distinct from that of the ordinary variety. Not only is the pain of neurotic origin, but also the distention is a result of paralysis of the muscular fibre of the gut. The processes in the gut may be disturbed as a result of the pneumonia. Colic may occur in breast-fed or in artificially fed infants. In the former it is not always possible to discover the exact cause. The breast milk may be abundant, of good color, and of correct composition, and still there may be very violent colicky pains. In artificially fed infants the cause of the colic may lie in the very nature of the food (cows' milk) and the difficulty of complete assimilation. Thus not only will an excess of proteids in the milk cause colic; the nature of the proteids of cows' milk, no matter how much they are diluted, will cause colic. An attack of colic is preceded by general uneasiness; the infant cries and cannot be quieted. The severe colicky pain is accompanied by sharp cries, the arms and lower extremities are drawn up, and the abdomen is rigid. After the passing of gas the infant is quieted and falls asleep quite exhausted. These attacks of colic deprive the infant of sleep; they may or may not be accompanied by tympanites. The movements are rarely normal, or may be normal for some days and then take on a curdy character or become greenish. Sometimes the colicky attacks are accompanied by a mild form of diarrhœa; the pain may be so severe as to cause convulsions.

Tympanites.

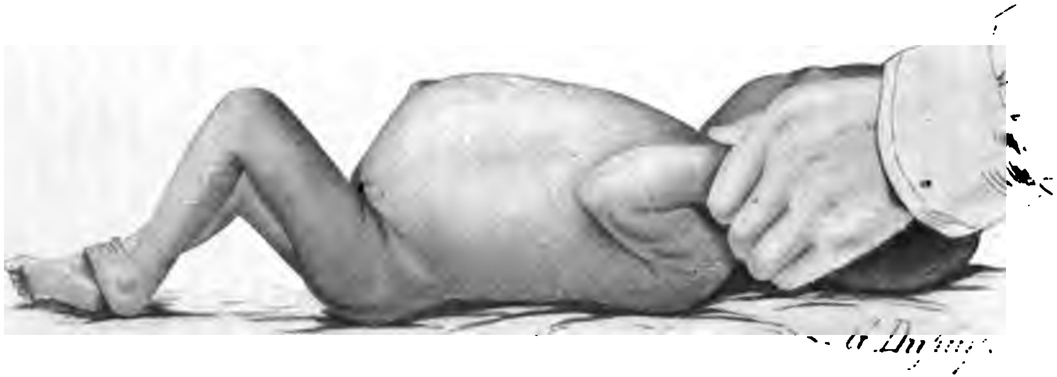
Tympanites is a condition of distention of the gut with gas, which may supervene in inflammatory states of the peritoneum. In such conditions (peritonitis, appendicitis) the paralysis of the muscular wall of the gut is the real cause of the distention. In other states, such as pneumonia, it may be the result of inefficient action of the diaphragm and of an enteric catarrh which sometimes accompanies that disease. In the newborn infant, tympanites is a result of an inherent muscular weakness of the intestinal wall. In colic due to imperfect assimilative processes in the gut, the tympanites is due to the formation of gases of which the intestine is unable to rid itself rapidly.

In pneumonia the tympanitic distention is sometimes extreme, causes great distress, and is frequently mistaken for peritonitis. In the forms of distention in the newborn infant the distress is not so great. In rachitis there is a state of tympanitic distention of the abdomen due not only to defective assimilative processes, but also to a lax condition of the muscle-fibre of the intestinal walls.

Treatment of Colic and Tympanites.—If the food of a bottle-fed infant is at fault, the modification of milk must be altered so that the proportion of the proteids may be lower. A reduction of proteid will not always remedy the condition; the proportion of sugar is sometimes at fault, especially in infants fed on condensed milk. Not more than 6 per cent. of sugar should be added to any milk modification. Some infants can take a large quantity of malt-sugar in their food and not suffer from colic. If a breast-fed infant suffers from colic, the hygiene of the nurse should be attended to. If after the taking of exercise and regulation of diet the colic persists and becomes a feature in the case, the wet-nurse should be changed.

The attack of colic is best combated by giving the infant an enema. In some cases a small amount of dilute hydrochloric acid and pepsin

PLATE XVII.



So-called Idiopathic Abdominal Distention or Tympanites in an Infant Nine Days Old. Recovery.

given three times daily will alleviate the symptoms. If in spite of all efforts an artificially fed baby suffers with colic and does not increase regularly in weight, it should be placed at the breast.

Dilatation of the Stomach.

Etiology.—Dilatation of the stomach may be due to mechanical causes, such as stenosis of the pylorus, resulting in overfilling of the stomach, with consequent dilatation; or it may be caused by muscular atony, such as is present in general atrophy or rachitis. In mechanical stenosis of the pylorus the muscular structures are intact at first; hypertrophy subsequently appears in the region of the pylorus, with secondary dilatation of the fundus of the stomach. An hour-glass distortion of the form of the stomach and, subsequent to this, a sausage-shaped dilatation of the organ result, the long diameter of the dilatation being in the long axis of the stomach. This last-named deformity is permanent.

The location of a dilated stomach in the child differs somewhat from that in the adult. The pylorus in the child lies deepest and near the umbilicus in the mid-line; the fundus lies transversely across the abdomen at the situation of the umbilicus; whereas in the vicinity of the border of the ribs it passes abruptly upward. The muscular coat of the stomach in these cases is thin and atrophic. If there is overloading of the stomach, or the ingestion of indigestible substances, the organ is not thoroughly emptied, and as a result there are fermentation and accumulation of food in the stomach. Muscular relaxation results, and then atrophy of an otherwise weak musculature. In athrepsia and rachitis the musculature of the stomach is primarily weak, and repeated attacks of dyspepsia with overloading result in dilatation.

The **symptoms** of dilatation of the stomach as a result of pyloric stenosis are described elsewhere. As a result of chronic dyspepsia and overloading of the stomach in younger children there are at first the ordinary symptoms of evanescent dyspepsia. There is vomiting after meals, and after a time this vomiting takes place after the food has accumulated in the stomach. With the attacks of vomiting there is loss of appetite, and finally an intolerance of all food, even in very small quantities. Constipation follows as a result of lack of appetite and the avoidance of food. Meteorism is present in some of these cases; whereas in others intestinal catarrh may alternate with the constipation.

In older children dilatation of the stomach results from repeated attacks of dyspepsia which extend over months. The development of the disease is slow. There are loss of appetite, a feeling of tension and overloading after meals; the odor of the breath is bad; the tongue is coated; children complain of headaches; the bowels are very irregular, sometimes constipated; and finally vomiting after meals sets in. The vomited matter contains not only particles of food, but sarcinæ and other species of bacteria. The reaction of the stomach-contents may be neutral or acid, the hydrochloric acid and propeptone may be increased or may vary on different days; lactic, butyric, and acetic acids may be present in the vomited matter as a result of fermentation.

The **physical signs** consist of persistent meteorism and tympanites. The abdomen is very much enlarged, and in some cases the stomach can be distinctly outlined, especially the greater curvature. If the child is examined lying on its back, with its knees raised and the pelvis supported with one hand while the other taps the abdomen sharply over the situation of the stomach, the distended organ will yield a so-called succussion sound, due to accumulated contents in the organ. In many cases a dilated colon may be mistaken for a dilated stomach. By means of gastrodiaphany the author has been able to mark out quite distinctly the greater curvature of the stomach.

The **prognosis** will vary according to the exciting cause. If the dilatation of the stomach is caused by congenital stenosis of the pylorus the prognosis is bad; if caused by repeated attacks of gastric dyspepsia the prognosis is more favorable. It is not as favorable in severely rachitic children, in whom there may be at the same time a progressive atrophy of the muscular tissue of the stomach.

The **treatment** of dilatation of the stomach in infants and children does not differ materially from the treatment of the same condition in the adult. In infants the quantity of solid food and fluids given at each meal is reduced to a minimum. The systematic washing of the stomach at intervals is indicated in these cases, as in older children and adults. With older children the amount of fluids is also limited. Soups are excluded and milk is peptonized. Bread, meat, and digestible substances are preferred to fluids. In these cases also the stomach is washed systematically.

The medical treatment of these cases consists in the administration of hydrochloric acid, pepsin, general hygiene, massage, faradization of the stomach in severe cases, as in the adult.

Ulcer of the Stomach.

Ulcer of the stomach may occur as a complication in sepsis of the newborn, in acute gastritis, and in tuberculosis. As a primary disease, this affection is very rare in infancy and childhood, although cases are reported in the literature as a complication of infectious diseases, such as scarlet fever, typhoid fever, measles, tuberculosis. Reimer records a case in a child three and a half years of age. Hibbard met a case in an infant four months of age. Rotch reports a case in an infant seven weeks old. It is rare, however, between, the ages of one and ten years. In 226 autopsies Brinton saw it twice. I have seen it at an autopsy in a case of empyema. It occurs in chlorotic girls toward the age of puberty, and is not a disease of infancy and childhood.

Hypertrophic Pyloric Stenosis.

(*Congenital Stenosis of the Pylorus; Congenital Hypertrophy of the Pylorus and Stomach-wall; Congenital Gastric Spasm.*)

Hypertrophic pyloric stenosis is a congenital condition which appears from a few days to several weeks (three months) after birth, and manifests itself in persistent vomiting. In a few instances several infants in the same family have been thus affected.

The first case of pyloric stenosis was described by Dr. Beardsley in the *Transactions of the New Haven Medical Society* (Osler).

The **etiology** of the affection is obscure. Since in the majority of the cases which have been carefully studied the infants were overfed or improperly fed, it is supposed that some irritant to the stomach is the exciting cause. Thomson, who has made careful studies of these cases, believes that the condition originates in intra-uterine life, and is due to the ingestion of liquor amnii. This fluid, by irritating the mucous membrane of the stomach, excites both that organ and the pylorus to overaction. Pfaundler, voicing the teachings of Escherich's school, denies that there is a true hypertrophy of the pylorus, and asserts that the condition during life is that of functional spasm. The post-mortem condition is due to toxic agonal contracture of the pylorus.

Morbid Anatomy.—The stomach and œsophagus have been found to be dilated in fully one-third of the reported cases. The mucous membrane shows the usual changes, such as the congestion which is seen in a stomach in which there have been functional disturbances. The mucous membrane of the pylorus is thrown into voluminous folds. The lumen has in some cases been found patent to a small probe, but fluids cannot be forced from the stomach through the pylorus (Thomson). The muscular fibres show characteristic change. The circular fibres are thickened and hypertrophied (Thomson). In Finkelstein's case the longitudinal fibres were also thus affected. Some deny this hypertrophy and contend that it is an agonal contraction.

Symptoms.—Infants in whom this condition is present are of normal weight and appearance when born. As a rule, the desire for food is greater than is normal. In the majority of cases the infants are allowed, on account of supposed insufficiency of the breast milk, to nurse an excessive length of time. It is soon noticed that there is vomiting of small quantities of milk after each nursing. After a few days all the food taken into the stomach is rejected, and the vomiting attacks increase in frequency. The vomited matter rarely contains bile-stained matter, though I have seen a case with bile-stained vomitus. At times the amount vomited is less than or about equal to the quantities of food ingested at the nursing. At intervals attacks of vomiting occur during which more food is rejected than has been taken at the preceding nursing. This proves that there is not only retention of food, but also lack of absorption by the mucous membrane of the stomach. The bowels are either constipated or very little in the form of fæces passes through the gut. In some cases a little fæcal matter resembling meconium is passed; whereas in other cases the stools are greenish, contain mucus and but very little fæcal matter. Toward the close the movements may contain mucus. In other cases the movements are scanty, but otherwise normal.

The infants rapidly lose weight; the abdomen has a characteristic appearance in all the cases I have met. The abdominal walls are lax, the coils of gut can be clearly made out, and the peristaltic movements are visible. In the epigastrium, just beneath the ribs, is a large coil, and to the right of this a constricting band and what appears to be another coil. These are in constant peristalsis. I have seen these coils in two cases, and in both they had been mistaken for the stomach. They were, in my opinion, coils of the transverse colon. In two of my cases I could on deep palpation in the median line, midway between the ensiform cartilage and the umbilicus, feel a resistant nodular mass, which may have been the contracted pylorus. I have not been able to map out a dilated stomach.

Prognosis.—If the vomiting is unrelieved, death occurs in from three weeks to six months. Cautley and Dent have reported 50 cases of pyloric stenosis, 19 of which died within three or four months. Of their own cases, of which they report 7, all died but 2, and these were operated by means of pyloroplasty, an operation which they advocate in preference to other operations. One infant weighed 8 pounds when operated on, and was eight weeks old; another weighed 9 pounds and 6 ounces and was six weeks old. On the other hand, all of the cases not operated on are not hopeless. Heubner has seen three cases which recovered. I have seen three cases in which the history, symptoms, and physical signs were undoubtedly those of congenital stenosis, and all recovered. One case gained to a remarkable degree in weight, another in three months contracted gastro-enteritis and subsequently a terminal intussusception and died unrelieved; the third recovered.

Treatment.—All mouth-feeding should be suspended. The infant is kept in the recumbent position, and the stomach washed out once; all binders and constricting clothing are removed from the abdomen. The infant is then fed by the rectum for one or more days with small quantities of albumin-water, or an ounce of somatose solution is introduced per rectum three or four times daily. This amount, although seemingly small, will if retained, sufficiently nourish the patient. The stomach is thus given complete rest. After two days teaspoonful doses of albumin-water are given by mouth every hour, and are supplemented by rectal enemata. If the vomiting has diminished or ceased, artificially fed infants are given by mouth a very dilute milk modification (0.5 of proteids, 2 of fat) in half-ounce doses, pasteurized or sterilized. The milk is given alternately with albumin-water. Milk is at first given only three times in the twenty-four hours alternately with albumin-water. If vomiting recurs, mouth-feeding is again suspended and rectal alimentation substituted. The infant is not moved, especially after being fed. In this tentative way, gradually increasing the quantity and strength of the milk (1.2 of proteids, 2.5 of fat), I have succeeded in three cases in effecting tolerance of food and cessation of vomiting.

Medicines are of little value in these cases. I have used small doses of bismuth subnitrate in combination with pepsin, to allay the irritability of the stomach.

Surgical Treatment.—Two surgical procedures have been proposed and attempted in these cases. The first method is that of opening the abdomen and establishing a communication between the stomach and gut by means of a Murphy button. This procedure, first suggested by Schwyzer, was carried out by Willy Meyer on the case of Meltzer, with fatal results. The second method is that adopted by Nicoll in the case of Ritchie. The abdomen was opened, an incision made near the pyloric end of the stomach, the pylorus forcibly dilated with forceps, and the wound closed. The infant recovered. I believe that with these weak infants every expedient should be tried before resorting to surgical intervention.

Acute Gastro-enteritis (including Cholera Infantum).

(*Summer Diarrhœa; Acute Gastro-enteric Infection.*)

Acute gastro-enteritis is a form of diarrhœa usually accompanied by gastric symptoms. It is prevalent in the summer, but may also occur during the winter months. Bottle-fed infants are more subject to the affection, although it occasionally attacks infants at the breast. In institutions epidemics of gastro-enteritis occur in breast-fed infants. In large cities more than one-half the deaths among infants under the age of twelve months are caused by summer diarrhœa. In Paris, Chaterinkoff found that of 20,000 children dying of gastro-intestinal disorders, fully three-fifths were bottle-fed. This high rate of the mortality of bottle-fed infants, as compared with that of breast-fed infants, is not alone due to the difference in the nature of the food; no matter how carefully it is handled before it reaches the infant, milk passes through many channels, and in each of these it is exposed to infection. The intense heat of summer also favors the increase of infectious agents.

Classification.—The various forms of acute gastro-intestinal infection may be divided into those whose source of infection lies outside the body (ectogenous) and those in which the elements of infection are pre-existent in the body (endogenous). This classification (Escherich) of the diarrhœa of infancy is both practical and in accordance with the results of recent study. In the first class are included the diarrhœas of toxic origin and cholera infantum; in the second are included the diarrhœas which are caused by varieties of bacteria pre-existent in the gut, but which, in the opinion of Booker, Escherich, and Marfan, may under favorable conditions increase to enormous numbers and become virulent, According to Booker, no one specific micro-organism is the essential cause of gastro-enteritis or acute summer diarrhœa. Escherich has shown that the coli group may under certain conditions become virulent. Of the bacteria which are found in certain forms of gastro-enteritis, the Streptococcus enteritidis seems to have attracted the greatest attention. Booker first insisted on the importance and peculiar rôle of this micro-organism. He found these streptococci in great numbers not only in the stools of infants suffering from acute summer diarrhœa, but also in the walls of the gut and in the various organs of the body. Escherich, Libman, and Hirsch have confirmed the results of Booker. Escherich regards the Streptococcus enteritidis as an ectogenous infection. The udder of the cow may be the source of this micro-organism. Marfan and Booker are also inclined to believe that streptococci are able under certain conditions to increase in number and virulence and that they are one of the endogenous forms of infection by a micro-organism normally present in the gut. Among the other bacteria found in enormous numbers in the movements of infants and children suffering from acute gastro-enteritis are the Bacillus pyocyaneus (Kossel and Baginsky), Proteus vulgarus (found by Booker in choleriform diarrhœa), and the proteolytic bacteria. The last class comprises peptonizing bacteria, such as the Bacillus subtilus, Bacillus mesentericus vulgatus, and Tyrotrix tenuis. These peptonizing bacteria are not found in the gut or stools of the breast-fed infant either when in good health or sick. We may thus classify all diarrhœas of acute gastro-enteritis as follows:

1. Those due to improper food, or the so-called mechanical irritative diarrhœas (Booker).
2. The infectious form of gastro-enteritis (endogenous and ectogenous). This class would include the toxic diarrhœas of some authors.

Not only the food and the bacteria, but also certain changes in the gut play an important rôle in acute gastro-enteritis.

Morbid Anatomy—Stomach and Intestines.—Booker has described a superficial loss of the epithelium of the stomach and gut, as a constant lesion in all fatal cases of gastro-enteritis. It may be intact in some places and destroyed or eroded in others. The mucous membrane of the jejunum and duodenum may show less denudation than other parts of the gut. The epithelial layer of the mucosa is infiltrated with leucocytes in diffuse areas or nests. The infiltration may push the epithelial layer upward. The mucosa itself is infiltrated with polynuclear and mononuclear leucocytes to a varying extent. The mucosa shows superficial or deep ulcerations involving the crypts or villi. Heubner has described a form of necrosis which chiefly affects the epithelial structure without involving the deep mucosa. This occurs in cholera infantum. Booker also describes a bronchitis and a form of bronchopneumonia which are quite constantly found in fatal cases of gastro-enteritis. Hemorrhages into the lung tissue are common.

In the **kidneys** there is necrosis of epithelium in the convoluted and irregular tubules (Booker).

The **liver** shows fatty degeneration and necrosis of the liver-cells.

The **lymph-nodes** show focal necrosis.

The Role of the Bacteria.—Booker has demonstrated that no bacteria are found in the mucosa of the intestine if the superficial epithelium is intact. If there is a lesion of continuity of the superficial layer, the bacteria invade the mucosa in large numbers. There is reason to believe that the toxins generated by the bacteria in the gut cause the superficial erosions and prepare the way for invasion of the lymph-channels and bloodvessels. Bacteria are not always found in the lesions, but as a rule the ulcerations of the mucosa show vast numbers. Booker found bacteria in cultures taken from the solid organs and blood, thus confirming what Czerny and Mozer found to be the case during life. The lungs especially showed large numbers of bacilli and cocci.

Symptoms—In the mild form of gastro-enteritis the infant is restless and cries at intervals because of colicky pains. It may previously have been in good health, but with the advance of these symptoms there will also be noticed a slight febrile movement and a disinclination to take the bottle or breast. Vomiting occurs after feeding, the rejected contents of the stomach being curdled and having a marked acid odor. In mild cases the vomiting is usually not severe. It may be repeated three or four times in the twenty-four hours. The movements are at first normal; they afterward become frequent and contain whitish curds or greenish and white curds, are more fluid than is normal, and may have a very offensive odor. In mild cases there may be only two or three such movements in the twenty-four hours or they may number six or more. Later, the fever also becomes more marked, the temperature sometimes mounting as high as 103° F. (39.4° C.). If the feeding is continued, the vomiting persists. The infant shows little or no prostration.

In severe cases the vomiting is marked from the outset. The infant not only vomits its regular food, but will also often vomit all fluid that is taken into the stomach. The diarrhœa is also more severe than in the mild forms. The movements are at first yellow or greenish and contain white curds, but as the disease advances they become more fluid, until in very severe cases only a greenish malodorous liquid containing small particles of mucus and fecal matter is voided. The infant has a febrile movement which varies from 101° to 103° F. (38.8° to 39.4° C.), and there is marked prostration. In the acute forms of gastro-enteritis there is considerable loss of weight; the infant becomes pale and languid, and the pulse is rapid and weak; the number of daily evacuations may reach twenty. In some cases the straining causes a descent of the lower part of the rectum, and the movements contain a slight amount of bloody mucus. The odor of the evacuation may not be offensive. If the patient improves, the symptoms retrograde —the vomiting becomes less frequent, the stools more fecal in character and less numerous, and the fever subsides. If, on the other hand, the symptoms progress, the movements not only continue frequent and fluid, but also blood and particles of mucus are mingled with the fecal matter. The vomiting may cease entirely. The infant loses in weight steadily; the movements are small and passed with tenesmus; the patient passes into the subacute stage of gastro-enteritis. In some cases there is colic; the infants are restless or pass into an apathetic condition. Little urine is passed, and in the majority of cases of mild or severe gastro-enteritis, albumin is present. It rarely amounts to more than a trace. In severe cases there are leucocytes and epithelial, hyaline, and blood-casts in the urine; sometimes in addition a few blood-cells are found.

In the subacute forms of gastro-enteritis which last for more than a week, bronchopneumonia may be a complication. This form of bronchopneumonia is described in the section on Pneumonia. In some cases it is of short duration, in others persistent. Bronchopneumonia with slowly resolving areas of consolidation in the lung is the type met with.

Course and Prognosis.—The prognosis of the mild forms of gastro-enteritis is good, if proper measures are adopted. The severe forms are exceedingly fatal in summer. The mortality varies with the environment. In the crowded tenements of large cities and in unhygienic surroundings the mortality is great, as is also the case in institutions and hospitals. In private practice the isolation of the patient and special nursing reduce the mortality to a minimum by preventing reinfection. Reinfection is caused by lack of care in handling the diapers and in preparing the food, by giving improper food, and by placing a number of cases in the same room. There can be no question that in hospitals patients are affected unfavorably by proximity to other patients suffering with the same disease. No matter how careful the nursing under such circumstances, reinfection cannot be prevented. Also, perfect cleanliness is not attainable in hospitals as in private practice.

Cholera Infantum.

Cholera infantum is the severest form of summer diarrhœa prevalent among infants. It is believed that it has a specific origin, but this has not as yet been demonstrated. Cholera infantum does not occur so frequently as has been hitherto supposed. Of hundreds of cases of gastro-enteritis of the acute variety which come under my care yearly, only a few can be called typical of this form of infectious diarrhœa. These cases occur for the most part in weakly bottle-fed infants. Breast-fed infants may occasionally be affected, especially in hospitals.

Symptoms.—The infants as a rule have been suffering from a mild diarrhœa. Following a slight febrile movement, vomiting and diarrhœa of a severe and exhausting character set in. The bowel movements are frequent, but contain very little fecal matter after the first few have been passed. They are at first greenish, afterward becoming watery, resembling barley-water; they contain but a few flocculi of mucus, and may not have much odor. The vomiting is incessant. First the stomach contents are vomited, and finally a greenish fluid. Within a few hours the infant is reduced to a condition of great prostration. The loss of weight is marked, even in the first twenty-four hours. The skin on the thighs is wrinkled.

The face and trunk are pale and the face is drawn. There is fever to a marked degree (101°–103° F., 38.3°–39.4° C.), and the pulse is rapid and thready. Toward the close the movements are passed involuntarily. The whole picture is that of a choleriform disease. As the fatal issue approaches the eyes become sunken and glassy, the fontanelle is depressed, and the mouth is open. The condition described elsewhere as hydrocephaloid sets in. Convulsions and a rise of temperature (105° to 107° F., 40.5° to 41.6° C.) precede the fatal issue.

Occurrence.—These severe choleriform diarrhœas resemble Asiatic cholera very closely, and should be sharply differentiated from severe forms of gastro-enteritis. They occur in bottle-fed infants under the age of two years, and chiefly in the months of July and August. Heat and infected food are the main etiological factors. A diarrhœa of a mild type is the forerunner in the majority of cases. These cases are not so frequent to-day as they were in the days when infants were fed with decomposed milk containing bacterial toxins. This form of diarrhœa must therefore be looked upon as a purely ectogenous infection.

Duration and Prognosis.—The prognosis in the majority of cases of cholera infantum is grave. The disease is an exceedingly fatal one, occurring as it does for the most part in infants fed on the bottle and whose general condition is poor. It lasts for from twenty-four hours to two or three days. The rapidity of the development of the symptoms and of the fatal results precludes the possibility of any complications other than those due to the great drain on the system. The condition of hydrocephaloid is hardly a complication; it is a terminal set of cerebral symptoms. Sclerema, mentioned by some authors, I have not met in true cholera infantum; it is seen in the terminal stage of acute forms of gastro-enteritis. This form of sclerema affects the thighs at the upper and inner part. It is described in the section devoted to that subject.

Kjelberg, Felsenthal, Bernard, Morse, and the author, found albumin and casts in the urine of children suffering from all forms of gastro-enteritis, acute and subacute, including cholera infantum.

Morse as well as the author found that the urine was concentrated and contained hyaline, granular, and epithelial casts, with leucocytes and blood and blood-casts. The albumin is rarely present to a marked degree. It is a trace or a distinct reaction. The urine is suppressed in severe cases, and lessened in quantity in others. In some cases of gastro-enteritis of the severe types there is slight œdema of the subcutaneous tissues, especially on the inner part of the thighs, the legs, and dorsum of the foot. We are not in a position to trace any close relationship between the general symptoms and the disturbances of the kidney. The toxæmia in this disease, causing as it does vomiting and nervous symptoms, masks the nephritic symptoms if they are present.

The **diagnosis** of acute gastro-enteritis is not difficult. There are, however, many infectious diseases, the onset of which closely resembles that of an attack of gastro-enteritis. Scarlet fever, for example, begins with vomiting, and in some cases with diarrhœa. There is a form of grippe which in its onset, with vomiting and diarrhœa, closely resembles an attack of gastro-enteric disease. In fact, these symptoms may persist in the course of the former affection.

The physician should not be satisfied with a history of gastro-enteric symptoms, but should carefully examine the skin, throat, and chest at every visit. In the severer forms of diarrhœa a small particle of the movement may be spread on a cover-glass and examined for an excessive number of streptococci. In mild forms of diarrhœa we should not fail to make a Widal test of the blood and a count of the leucocytes, to eliminate the possibility of typhoid fever. This will especially be indicated in cases in which there is enlargement of the spleen.

Treatment of Acute Gastro-enteritis and Cholera Infantum.—Prophylaxis.—The nursing bottles when emptied by the infant should be filled with a saturated solution of sodium bicarbonate, allowed to stand for a few hours, and then carefully washed inside and out with a bristle brush. The nipples should be sterilized daily. The nurse or mother, after attending to the diapers of the infant, should carefully cleanse the hands before feeding the baby. The milk should be diluted as directed in the section on Infant Feeding, pasteurized or sterilized, and then kept on ice until needed. It should be obtained from a dairy in which cleanliness of the utensils and in the milking of the cows is observed in all details. The milk should be fresh and delivered for modification within a few hours of the milking-time. The nursings should be conducted at stated intervals. If there is a residue in the nursing bottle, it should not be utilized for a subsequent nursing. The infant is given a full bath daily. By attending to all these details, infection of the food and of the infant may be avoided. With breast-fed infants prophylaxis is of great importance. A baby at the breast should be fed at regular intervals. The breast-nipples should be washed with a saturated solution of boric acid before and after nursing. The baby should not be allowed to nurse a breast with a fissured nipple. The milk of such a breast is pumped off, and an attempt is made to heal the nipple in the manner elsewhere described. If there is caking of the breast, the condition should be remedied before the infant is allowed to nurse. Abundance of fresh air and bathing are indicated in these infants as in bottle-fed infants.

Sick Infants.—As soon as a baby shows signs of even mild dyspepsia or gastro-enteritis the milk should be discontinued, a simple cathartic given, and the infant kept for twenty-four hours on a solution of egg-albumin. Vomiting which has occurred only once or twice does not call for active treatment, as it will disappear as soon as the milk is discontinued. After the bowels have moved, if the infant shows no exacerbation of symptoms feeding should be resumed cautiously. In this way a severe gastro-enteritis can be averted. If the food is not suitable, causing signs of dyspepsia such as colic, it should be changed if possible, else severer symptoms may result. If in spite of all precautions an attack of gastro-enteritis develops, the patient should be treated on the following lines:

1. The food is stopped and another of a safe character substituted.
2. The toxins are eliminated and the strengh of the patient supported by the so-called mechanical methods.
3. Drugs are used to abate the symptoms and support the strength of the patient.

The milk, whether of the breast or bottle, is discontinued. The infant is given a solution of albumin-water, acorn-cocoa, or beef-juice expressed and diluted with barley-water. A baby can be kept for days upon these mixtures without any danger of reducing the strength.

According to Czerny, 100 c.c. of breast milk are equivalent to 61 calories; 100 c.c. of the white of egg are equal to 75.1 calories. The white of one egg weighs about 30 grammes; therefore the white of an egg is equal to about 25 calories. It is digestible, and is well borne by infants. Albumin-water may be used alternately with the solution of acorn-cocoa or beef-juice and barley-water. To older children who are suffering from gastro-enteritis we may sometimes have difficulty in administering albumin-water or acorn-cocoa. Under such conditions I frequently resort to a dextrinized gruel or the so-called Liebig's soup mixture which Keller advised. I find that after the acute symptoms are past, infants and children who refuse every other form of food will take this with eagerness. It may be given while the diarrhœa is still in progress, but should not be given until the vomiting has ceased.

The cathartic given at the onset should be castor oil or calomel, ½ grain (0.03) doses twice or three times a day. Infants who are vomiting are given calomel in preference to castor oil.

The Vomiting.—If the vomiting is not severe and the case is under treatment from the onset, it is best not to wash out the stomach at once. It often happens that the vomiting ceases as soon as the regular food is stopped. If, however, the vomiting persists for twenty-four hours, we proceed to wash out the stomach once. If the vomiting continues after this, it is either toxic or may in rare cases be due to some other causes. As a rule, it ceases after one irrigation of the stomach.

The **diarrhœa** is controlled by irrigation of the gut. The rectum and gut are washed out in those cases in which the diarrhœa is not only persistent, but progressive. The object in washing out the lower bowel in any form of acute gastro-enteritis is twofold: (*a*) to remove any residue of feces that may have collected in the lower bowel and rectum, and to stimulate peristalsis and thereby favor evacuation from above; (*b*) to stimulate the heart and add to the body an amount of normal solution to compensate for the drain caused by the diarrhœa. The Cantani normal salt solution is utilized in the manner described on page 68.

The rectal enemata are given under a pressure obtained by an elevation of at most two feet from the bed. A temperature of 107° to 110° F. (40.5° to 43.3° C.) is the best and most stimulating in these cases. Fully a quart of water is thrown into the rectum in half-pint portions. As the half-pint flows in, the funnel on the rectal tube is disconnected and the contents of the bowel are allowed to escape. Another portion is then allowed to flow into the bowel. The water will sometimes escape alongside of the tube. This is rather a favorable sign, being significant of the contractile powers of the gut and abdominal walls. Only two enemata daily are necessary, even in severe cases of acute gastro-enteritis. As the diarrhœa and symptoms subside we reduce the number of enemata to one, finally discontinuing them entirely as the infant improves. It sometimes happens that after a few days the enemata are followed by movements containing blood and mucus, the tenesmus being aggravated. In these exceptional cases an enema must be given only every other day, and the effect on the rectal discharges watched. By stopping the enemata altogether it can be determined whether the discharges of mucus and blood are caused by the therapy or the disease.

Hypodermoclysis.—The injection of normal salt solution under the skin is indicated only in the severe cases of acute gastro-enteritis in which, as in cholera infantum, the course of the disease is rapid and the prostration extreme. Personal experience rather discourages the employment of large injections by this method. I have seen two cases of infection by the Bacillus capsulatus aërogenes (Welch) following hypodermoclysis. These occurred through the use of saline solution evidently insufficiently sterilized, and which had probably been allowed to stand before being used. In a third case hemorrhages over large areas occurred at the point of the injection of the salt solution. These injections are also very painful. Because of these dangers and disadvantages the subcutaneous injections of salt solutions should be utilized as a last resource in desperate cases. Small rather than large amounts of fluid should be injected subcutaneously, as was advised on page 36. The salt solution for the hypodermoclysis is that of Cantani. It should be sterilized at a temperature of 212° F. (100° C.) for at least an hour, to kill sporulated bacteria if possible. A very fine hypodermic needle is used in the manner described on page 62.

Baths.—In all cases of acute gastro-enteritis, whether with or without elevation of temperature, the benefit obtained from warm baths cannot be overestimated. In cases of great prostration a bath at 108° F. (42.2° C.) for five minutes is stimulating to the nervous centres and is followed in many cases by diminution of the apathy and an apparent reduction of the effects of toxæmia. If the temperature rises above 103° F. (39.4° C.), sponging with water at 80°–85° F. (26.6°–29.4° C.) is all that is needed. This should not be done oftener than once in every three hours.

Alcohol.—Of late years, whiskey is given less and less in cases of acute gastro-enteritis. In these cases there is a special intolerance of the stomach and also of the economy to whiskey. Infants after taking it for twenty-four hours will become stupid, apathetic, and exhibit a constant retching if they do not vomit. This appears to be due more to the effect of the alcohol locally on the stomach and also systemically than to toxæmia of the disease. I therefore deprecate the use of alcohol except in extreme cases, when whiskey is given in small doses at short intervals.

Strychnine is useful for its stimulating effect on the heart. Grain $\frac{1}{300}$ (0.0002) is given to an infant of six months, and grain $\frac{1}{200}$ (0.0003) to older infants every three hours.

Atropine, lately advised as a cardiac stimulant in these cases, especially in cholera infantum, is a dangerous drug, and should not be employed. I have seen grain $\frac{1}{150}$ (0.0004) give rise to constant tremulous and convulsive twitching.

Resorcin.—If the vomiting is constant, grain $\frac{1}{8}$ (0.008) of resorcin given every three hours is a safe and very useful remedy. It should never be used in larger doses nor at shorter intervals.

Bismuth in the form of the subcarbonate is the only drug useful in allaying the vomiting and the tenesmus of the bowel. Grains ij or iij (0.12 or 0.18) are given in powder form every two or three hours.

Opium in any form has fallen into disuse. In the severe cases it is dangerous, and may increase the prostration; in the milder cases its use is justifiable only if the colicky pains are excessive. The milder preparations such as the wine and the camphorated tincture are of value, because they can be given in graduated doses, and the effects determined more exactly than can be done with the stronger preparations.

Salol in grain $\frac{1}{2}$ (0.03) doses every three hours may be combined with the bismuth to allay the colicky pains.

Tannigen is a useful drug in the chronic forms of intestinal disease, but an irritant in the acute forms.

Colic has been mentioned so often that a few words as to the treatment may not be out of place. Passing of the rectal tube rarely relieves it. A small rectal enema has been found to be a very effective remedy.

As the symptoms improve care should be taken not to return to a milk diet too quickly. The milk is given in dilutions and is sterilized carefully. Infants in an enfeebled condition as a rule bear this form of milk best, since it is not apt to be irritating to the gut. When the danger is past any form of milk may be given—raw, pasteurized, or sterilized—care being taken that all the precautions as to freshness, cleanliness, and proper preparation are observed. I have mentioned the fact that before returning to dilutions of milk the exhibition of dextrinized gruels has been successful with very weak infants. The malt, the cereal, and the milk acted upon by the ferment contained in these mixtures are all easily digestible and assimilable, and promote increase of weight. As a matter of course, the effect of the gruel mixture on the stomach and gut should be carefully studied.

Whatever methods are employed in the treatment of acute gastro-enteritis, it is necessary to avoid the error of overtreatment. It should be remembered that hours of rest do more than hours of treatment. Three-hour intervals should elapse between the application of remedial measures. Fresh air in the room or a sojourn of a few hours in the open with absolute quiet, is of the greatest value in these cases.

Infantile Atrophy.

(*Marasmus; Athrepsia* (Parrot).)

Atrophy may follow or complicate congenital syphilis or any subacute or chronic disease of the gut. Infantile atrophy, or the athrepsia of Parrot, is a condition due to a faulty operation of the assimilative processes in the gut.

The **etiology** of chronic atrophy is unknown. Keller, who has studied this condition extensively, believes that in infants and children suffering from gastro-enteric catarrh there is an excessive production in the gut of acids which under normal conditions are neutralized. In disturbed conditions, instead of urea, ammonia is excreted in the urine. The formation of the ammonia entails a drain on the economy—hence the emaciation. Excess of albuminoids and fats in the food favors overproduction of acids in the gut. In marasmus there is an acid intoxication of the economy, originating in the gut. There is no doubt that under unhygienic conditions and in overcrowded hospitals infection of one patient by another may take place. No satisfactory explanation of the manner in which such infection occurs has as yet been advanced. Heubner has surmised that the excreta of one patient in some way contaminates the food of another by transmission through the nursing personnel. This is true in a certain number of cases, but not in all. Cases of marasmus seen in private practice show progressive emaciation in spite of the fact that the utmost care is exercised in the preparation of food and the strict maintenance of hygienic conditions.

Morbid Anatomy.—The body is much emaciated; the skin hangs in folds on the extremities, and presents hemorrhages and petechiæ. The lungs may show atelectatic areas or may be the seat of bronchopneumonia. The heart is small and the muscle-fibre pale. In many cases the stomach is dilated and the mucous membrane pale. The small intestine shows few changes. The Peyer's patches may be slightly raised and show the so-called shaven-beard appearance. The follicles of the colon may be slightly prominent. The microscopical changes in the gut are not characteristic. In some places the follicles are the seat of catarrhal inflammation. Both in the stomach and the intestines there are patches where there is an absence of glandular tissue; in its place is a newly formed connective tissue composed of round and spindle-shaped cells. The villi of the gut have disappeared. The whole mucosa is thinner than is normal (Baginsky). On the other hand, these changes may not be marked. The liver is fatty and may be enlarged. The spleen is small. The kidneys may be pale, especially in the cortex, and may be the seat of parenchymatous degeneration. The lymph-nodes of the mesentery may be enlarged.

Symptoms.—Cases of gastro-intestinal atrophy are seen among the better classes, but, as a rule, they form a large contingent of dispensary cases exposed to unhygienic surroundings, and often improperly fed. The infant may have been small at birth or prematurely born. In some cases, especially in families in good circumstances, the baby may have been put at the breast and have done well up to the time when, for some reason, it was put on condensed milk or milk which had been carefully modified. In other cases the infant may from the outset have been fed on the bottle with good results up to a certain point, when the weight became stationary and the infant retrograded and showed signs of atrophy. The atrophic course having once begun, the symptoms vary little. There is no gain in weight. The skin, especially about the inner parts of the thighs, becomes wrinkled, and the subcutaneous adipose tissue diminishes in amount. The extremities lose their normal plumpness. The face has a pinched appearance. The chest is emaciated and the ribs show plainly. The fontanelles, if still open, may be somewhat depressed. Over the buccinator muscles is seen a small cushion of fat, the so-called "sucking pads," which remains when all the other fat has disappeared. This gives to the face a peculiar appearance that is typical of cases of atrophy. If the infant is not well taken care of, erosions and aphthæ are seen on the mucous membrane of the mouth and gums, and there may also be sprue. Even in well-cared-for infants the buttocks may be slightly eroded. If the infant has been neglected, there is marked intertrigo. The buttocks are emaciated and the tuber ischii show prominently. During the progressive emaciation the infants have constant gastro-enteric disturbances. There are colic and at times constipation, or diarrhœal movements alternating with constipation. The movements of the same infant vary greatly. They may be greenish with white curds, are sometimes exceedingly offensive, and at other times may be normal and alternate with slightly diarrhœal movements. The temperature is normal or slightly subnormal. During exacerbations of the intestinal disturbances, it may rise slightly. These disturbances of the gut do not seem to be influenced by changes in the diet. The infants in many cases finally lose all desire for food. Others drink with avidity, but do not assimilate the food taken. If untreated, these infants emaciate until they are reduced to skin and bone. They grow exceedingly weak, and die with some intercurrent infection, such as pneumonia, tuberculosis, or infectious disease.

Treatment.—If an infant suffering with chronic atrophy comes under treatment at from the third to the fifth month, the proper procedure is to place it on good breast milk. If this is not feasible and the bottle is the only resort, the task is more difficult. In rare cases carefully modified cows' milk (with the formula of proteids, 1.2; fat, 2.5; sugar, 6) will give the desired results if the infant has not previously had a correct diet, but has been given a proprietary food or a condensed milk formula. In my experience in ambulatory cases, no milk formulæ of any kind have been universally successful. I have found that many of these cases if put on a gruel and milk diet, according to the method described by Keller, and detailed elsewhere in this work, do well: the weight increases, the colic disappears, and the character of the stools improves. After the weight reaches a certain point the milk gruel is discontinued and the infants continue to do well on an ordinary milk formula. In certain cases of marantic infants of the age of twelve months this method has been very successful, especially with those whose distaste for the ordinary milk foods gradually increased. It is necessary to study out the method of feeding which seems likely to be best adapted to the individual case. Greater success can be attained in private practice than in hospitals. With the feeding, the general hygiene of the infant should receive attention. Daily baths with sea-salt and open-air life are especially indicated.

In infantile atrophy the medical and mechanical treatment are of less importance than the selection of proper food. For this reason we should not seek to multiply remedies. The movements of the bowels in some cases have an exceedingly fetid odor. The treatment is begun with the administration of brisk cathartics, such as castor oil. The bowel is then washed out once a day in the same manner as in gastro-enteritis until the character of the movements has improved. If there is a tendency to diarrhœa, tannigen, with or

without bismuth, may be given three or four times daily. If there is any great amount of gas generated in the stomach, a very small dose of dilute hydrochloric acid and pepsin should be given daily after a feeding.

Acute and Subacute Enterocolitis.

(*Enteritis Follicularis; Enteric Catarrh.*)

Enterocolitis is peculiarly a diarrhœal disease of infancy and early childhood. It was formerly classified as a form of dysentery, because in these cases the movements are tinged with blood and contain mucus. The cases are, however, really of a milder type, and present many symptoms foreign to true dysentery.

Etiology.—In many of its features this affection resembles acute and subacute gastro-enteritis. It is prevalent during the summer months. It occurs in infants after the first year of life, and may be primary or follow an ordinary dyspeptic diarrhœa, an exanthema, pertussis, or bronchopneumonia. Booker has described the great number of streptococci found in certain of these cases. Finkelstein and Escherich and his pupils have confirmed these results, and have in addition presented the view that these diarrhœas are infectious, and may be caused by bacteria of the coli group. The bacteria may be introduced from without, or the coli organism in the gut under certain conditions may become virulent. With reference to their origin, these cases may be considered as bearing a relationship to cases of true dysentery, from which with our present imperfect knowledge it is not always possible to distinguish them.

Morbid Anatomy.—The mucous membrane is hyperæmic and swollen; in cases of long duration the mucosa is infiltrated with small round cells. The follicles of the gut are enlarged and elevated above the surface of the mucous membrane. The Peyer's patches are enlarged and surrounded by a zone of hyperæmia. The villi show desquamated epithelium and infiltration of the walls with small round cells. The follicles are swollen, and at the surface may burst and present follicular ulcers. The epithelium of the gut may be lacking in places.

Symptoms.—In the beginning there are fever and slight vomiting. The movements are fluid, greenish, and have a disagreeable odor, contain mucus, and are streaked with blood. They may number ten or twelve in twenty-four hours. Straining at times accompanies the movement. As a rule the infant is pale and prostrated. The character of the movements is unchanged for from a few days to two or three weeks, when improvement begins and recovery ensues. On the other hand, in protracted cases the infant may develop a bronchopneumonia in one or both lungs, but may even then recover under good management. The picture thus resembles that of a mild dysentery, but the subjects are younger, and there is in a number of cases a history of antecedent intestinal disturbance of extensive duration.

The **treatment** should be carried out on the same lines as in acute gastro-enteritis. Caution should be exercised in returning to a diet composed exclusively of milk. While in true dysentery in older children I advise the administration of milk sterilized in some form, in younger infants such a procedure would be unwise. I keep these infants on a diet devoid of milk, such as beef-juice and barley-water, albumin-water or solution of acorn-cocoa, as long as possible. As the character of the movements improves the infants are put on a dilution of albumin-water and milk or cocoa and milk, or, what is far preferable, dextrinized gruel and milk. The amount of milk in the dextrinized mixture is gradually increased until the quantities appropriate to the age of the infant are given.

Dysentery.

(*Ileocolitis; Colitis Contagiosa; Coli Colitis; Enteritis Follicularis; Enterocolitis.*)

Dysentery is an acute infectious diarrhœal affection of the intestine. In the United States it occurs both sporadically and in localized epidemics. It is endemic in the tropics, where the etiology is somewhat different from that in our climate. The protozöon infection (amœbic) seems, according to Kartullis, to be characteristic of the tropical form. Although amœbic dysentery is occasionally seen here sporadically and in cases of persons recently returned from the tropics, it is not the form which commonly occurs in infants and children. The form to which these patients are liable is seen during July, August, and September, and late in the autumn. It may affect nurslings who are fed artificially, but most often occurs in children who are on a mixed diet. Escherich has described epidemics of limited character in private families and hospitals. I have met this form of dysentery in sporadic cases or small local outbreaks, and have also seen outbreaks at seaside resorts among children of from two to four years of age who had partaken of drinking-water which had been rendered unfit for use by contamination.

Etiology.—The essential cause of dysentery or ileocolitis is now recognized to be bacterial. It is due in certain cases to the introduction of bacteria of the coli group into the gut from without (Escherich). Maggiora, Celli, and others have described coli bacteria in the stools in epidemic dysentery. These authors have shown that these bacilli, which resemble the Bacterium coli of Escherich, may cause hemorrhagic colitis in lower animals. French writers think that the coli group existent in the gut may under abnormal conditions of intestinal disturbance assume a virulence not normal to them. Escherich, on the other hand, has endeavored to show that bacteria of the coli group, if introduced into the gut from without, either in the drinking-water or in food, may become very virulent.

Shiga, in 1897 and 1898, isolated a bacillus from the fæces of a number of cases of dysentery occurring in Japan. He discovered also that the blood-serum of the persons afflicted caused a clumping of the bacillus isolated when mixed with cultures of the latter in the proper dilutions. These cases of dysentery cited by Shiga did not include the amœbic variety. The characteristics of the bacillus isolated from these cases closely resembled those of the bacillus of typhoid fever, except that it was not motile and did not agglutinate with the blood-serum, as in the cases of typhoid fever.

In 1902 Duval and Bassett studied 53 cases of diarrhœa of the dysenteric type, and obtained cultures of the bacillus of Shiga in 42 of the cases investigated. Since then a number of investigators have studied the dysenteries of children in local epidemics, and have substantiated the work of Duval and Bassett, inasmuch as they are unanimous in attributing the dysenteric diarrhœas of infants and children as being due to this bacillus. The class of cases in which this bacillus is found is for the most part that in which the stools either show blood and mucus or mucus alone in considerable quantities. This classification does not include, as at first supposed, the cases of ordinary summer diarrhœa due to intestinal infection.

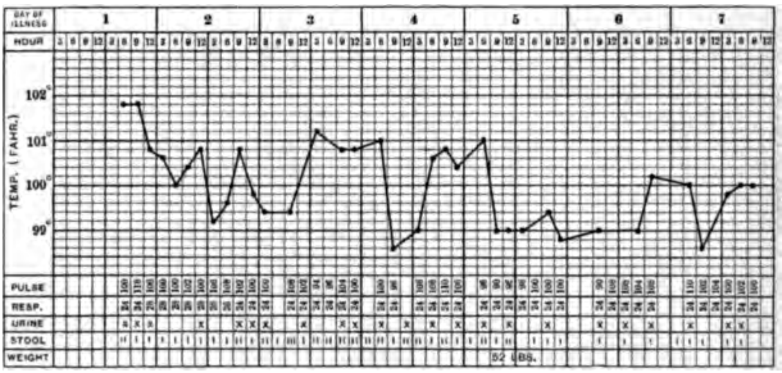
Morbid Anatomy.—Dysentery may affect different sections of the gut at the same time, the rectal or sigmoid flexure alone, the ascending colon, the transverse or the descending colon only. In rare cases the disease may pass beyond the ileocæcal valve and involve the lower part of the ileum. There are two forms which may be present separately or simultaneously in the same gut—the catarrhal and the necrotic form.

In the milder catarrhal form of dysentery the mucous membrane is hyperæmic and swollen, and the summits of the intestinal folds are studded with hemorrhages in small foci or streaks. The submucosa is infiltrated with small round cells and the vessels filled with blood. The epithelium of the follicles is swollen and proliferated, and there is infiltration of the surrounding connective tissue with round cells. In severe forms the surface of the mucous membrane is covered with mucus containing leucocytes and blood-cells. The follicles are elevated above the surface. In other cases the intestine is studded with ulcerations which mark the necrotic follicles. The ulcerations reach to the muscularis mucosæ. If the process extends to the small gut, the Peyer's patches are swollen and surrounded by a hyperæmic zone.

If the disease has advanced to the necrotic stage, the mucosa is thickened and infiltrated with round cells. There are areas of loss of tissue which extend deep to the muscular coat (gangrene). The mucous membrane is covered with a grayish exudate of a pseudomembranous character. In severe cases large areas of the mucous membrane may necrose and be cast off. The necrotic areas show an abundant invasion of bacteria of the coli type, in scattered masses or zöoglœa. The lymph-nodes of the mesentery are swollen; the spleen may be enlarged; the kidneys may show degenerative changes, and the lungs may be the seat of bronchopneumonia.

The **symptoms** of dysentery in infants and children closely resemble those seen in the adult subject. The onset may follow some indiscretion of diet or be entirely independent of any such error. There may be a preceding headache, and there is, as a rule, some fever. Abdominal pain is the first symptom until diarrhœa sets in. The diarrhœa at first resembles an ordinary dyspeptic diarrhœa, but in a few hours or after one or two movements, it assumes the characteristics which mark it as specific. The patient passes stools which are fluid and contain mucus mixed with blood and shreds of tissue, and which may have an offensive odor. They are passed with much abdominal pain and rectal tenesmus. If the abdominal pain is severe, there are vomiting and great prostration. As many as twenty to thirty small bloody mucoid movements may be passed daily. The fever varies in intensity. In mild cases the temperature may range from 101° to 102° F. (38° to 38.5° C.) (Fig. 97); in severe ones it may reach 104° (40° C.) (Fig. 98). If the disease persists beyond a few days, there is rapid emaciation and the abdomen becomes sunken and board-like. In some cases palpation in the

FIG. 97.

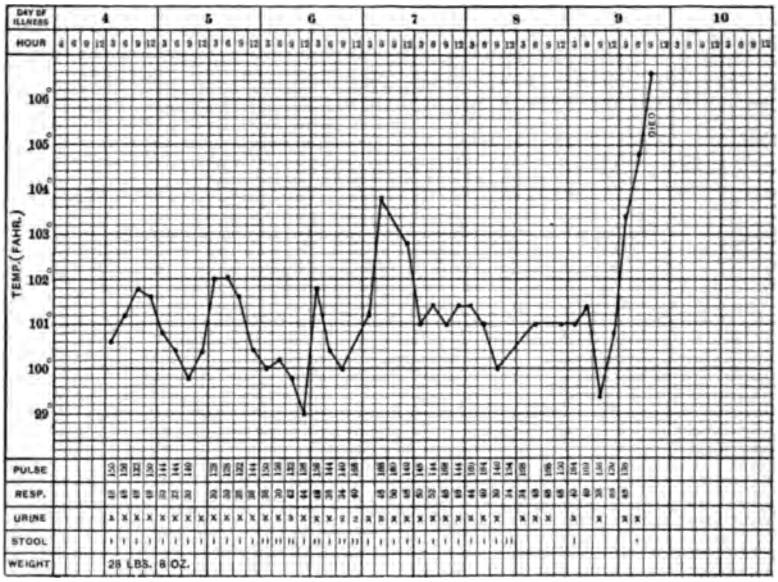


Dysentery of ordinary severity. First week of illness. Duration three weeks; recovery. Boy, seven years of age.

region of the cæcum and ascending colon may detect the contracted, thickened gut. In one case of the necrotic type, I could during life mark out the cæcum and ascending colon as a contracted, thickened tube. In protracted cases the spleen becomes enlarged and the tongue dry and coated, in this respect resembling the condition seen in typhoid fever. Multiple hemorrhages may appear under the skin. The urine contains albumin, and in some cases hyaline and epithelial casts.

Course.—The fulminating cases run their course in a few days with high fever, terminating in death. Other cases may be comparatively mild and last only a few days or a week. In such cases there may be recurrences. In other cases the disease runs a course of from three to six weeks. After this period, from time to time, blood, evidently derived from bleeding ulcers in process of repair, may appear in the movements. The movements gradually become formed and fecal in character, and the patient recovers. In cases which have come under my care in hospital service, the disease ran a moderately severe course until the seventh or eighth day. The fever, however, remained high and delirium set in on the ninth day. The appearance of the patient becomes septic, sopor supervened, and the

FIG. 98.



Necrotic colitis; fatal, in a girl six years of age.

urine and feces were passed involuntarily. Death took place on the thirteenth day. In other cases of a severe necrotic type death took place at the end of a week.

Complications.—The most dangerous complication is perforation and general peritonitis. Periproctitic abscess may occur, with subsequent fistula. In septic cases, abscess of the liver and spleen have been observed. Hemorrhages may occur under the skin late in the disease. In all of my cases these were quite extensive, but recovery nevertheless took place. In one fatal case I noted metastatic parotitis. Some authors have recorded arthritis as a complication; as a rule it retrogrades and recovery takes place.

The **prognosis** varies with the severity of the case. The mortality ranges from 30 to 40 per cent. The croupous or necrotic cases are very fatal. With good management the mild cases give a favorable prognosis. The severity of the infection and the prevalence of an epidemic will influence the course of the affection.

Treatment.—**Prophylaxis.**—The movements are not only infectious, but may also communicate the disease to others if a particle is introduced into the gut. The hands of the patient and his body should be kept scrupulously clean to avoid reinfection. The movements should be disinfected in the same manner as those of a patient suffering with typhoid fever. The hands of the nurse should be scrupulously cleansed and washed in an antiseptic solution.

The patient is given a cathartic, preferably castor oil, as the initial step of treatment. In this way all irritating food particles and residual feces are cleared from the gut. All food, even milk, is withheld at first. The patient for the first twenty-four hours is given a solution of egg-albumin, acorn-cocoa, beef-juice broths, or expressed beef-juice and barley-water in equal parts. The following are the lines along which the later management of these cases should proceed:

a. An absolutely non-irritating and easily assimilable food is given.

b. The pain and tenderness are relieved with drugs, the diarrhœa being also partially controlled in this manner.

c. The rectum is irrigated.

d. After a day or two, during which the patient has been fed upon albumin-water, expressed beef-juice, and barley-water or acorn-cocoa solutions, sterilized or pasteurized milk is substituted. In these cases, as in typhoid fever, the patients are given during twenty-four hours, two or more quarts of milk sterilized at 212° F. (100° C.) or pasteurized at 164° F. (73° C.). I wait until the severely acute symptoms have subsided before placing these patients on a milk diet.

At best, milk leaves a large residue in the gut, and in the acute stage of the disease the coagulum may in a mechanical way irritate the acutely inflamed walls. Pasteurized and sterilized milk is well borne in the later stages of the affection. Milk in a raw state, no matter how good, will sometimes tend to aggravate the acute symptoms. Pain and tenesmus are relieved by the exhibition of Dover's powder, grains ½ to ij (0.03 to 0.12), every two hours according to the age of the infant or child. Codeine sulphate, grain ⅙ to ¼ (0.01 to 0.015), according to the age of the patient, is preferable to morphine or tincture of opium. The administration of powdered ipecacuanha will be found very useful in certain cases. In others the vomiting rather interferes with its administration; grains j to ij or iij (0.06 to 0.12 or 0.2) every two or three hours are indicated. It may be combined with bismuth subcarbonate, grain v (0.3) every three hours.

In older children this mode of treatment has lately given good results. I have had no experience with the administration of lead salts. In the acute cases the internal administration of preparations, such as tannigen, is irritating.

Rectal enemata should be employed with care in the treatment of colitis or dysentery. Unless caution is exercised, their use is in many cases followed by an exacerbation or perpetuation of symptoms. The most useful form of enema is the warm (108°–110° F., 42.2°–43.3° C.) saline (Cantani) solution. Fully a quart of fluid is allowed to flow into the gut. The greater part of it returns, but I believe that if a portion of this solution is retained it acts in the manner of enteroclysis and supports the patient. These enemata are given three times in the twenty-four hours, for a day or two; they are subsequently given twice a day, and finally, as the symptoms subside, only once a day. I have never been able to convince myself that silver nitrate (1 : 1000) or tannic acid added to the enemata is of value. On the contrary, I believe that in cases in the acute stage these medicated enemata are distinctly irritating. In the later stages of the disease, small quantities of fluid blood are passed with the fecal movements, tenesmus being present; small enemata of silver nitrate (1 : 1000) given low down twice daily cause cessation of the bleeding which is due to the presence of ulcers low down in the rectum. In the subacute stage, the enemata will often be followed by an exacerbation of bloody mucous passages. Under these conditions it is well to discontinue the enemata and to watch the results of the suspension of local treatment.

Amœbic Dysentery.

Amœbic dysentery is not, strictly speaking, a disease of infancy and childhood. It is caused by the Amœbæ coli of Lösch. Of 35 cases reported by Harris, 4 were under ten years of age. Amberg has recently published 5 additional cases. The etiological factor is the Amœbæ coli, which are found in large numbers in the movements. With the amœba, Charcot-Leyden crystals are found in most cases. The cases published by Amberg were of a mild type, and seemed in no way to differ in symptomatology from the form of the disease seen in the adult subject. There were diarrhœa of a bloody character, tenesmus, and in some cases fever and prostration. As many as from four to six movements containing blood and mucus, and microscopically eosinophile cells, were passed in twenty-four hours.

The diagnosis is made from the presence of the amœbæ in the movements. Bloody passages containing Charcot-Leyden crystals should cause the physician to entertain a suspicion of the presence of this affection.

Other amœbæ, such as the Monocercomonas hominis (Grassi), have been found in the movements of infants suffering from diarrhœa. Epstein describes an epidemic of diarrhœa in which the monocercomonas abounded in the movements. He thinks that in this epidemic the diarrhœa was caused by well-water which contained the amœbæ. I have found the Monocercomonas hominis in the movements of infants who were suffering from diarrhœa, but also of those whose bowels were not in an abnormal condition. The rôle of the monocercomonas as an etiological factor in the causation of these diarrhœas is not understood. It is doubtful whether they have any causal connection with the diarrhœa.

Constipation in Infants and Children.

Constipation may be classified as congenital and acquired.

Congenital constipation is noticed immediately after birth, or in the days subsequent to it. The causes of congenital constipation are generally an absence of the anus or its occlusion by a thin membrane, or by a thick, hard membrane resembling the skin; or there may be an anus and a shallow or deep *cul-de-sac* leading from the anus for some distance into the rectum, or this may be occluded at a varying distance from the external orifice. The rectum may be occluded by one or several membranes. Its walls may be thickened, so that meconium or fæces cannot pass; or its walls may be agglutinated. The rectum, as has been stated, may end at some distance from the anus in a blind *cul-de-sac*, and from this point upward the rectum may either exist in its normal calibre, or may be simply indicated by a fibrous cord; in other words, there may be a congenital absence of the rectum. The rectum may end in a preternatural opening into the bladder, the urethra or vagina, or may, by a common opening, a sort of cloaca, terminate in the perineum through the urethra or vagina. In such cases there is scarcely constipation, but rather a difficulty in voiding the fæces. There may be, as has been intimated, partial or complete absence of the rectum or colon; or a large part of the larger bowel may be absent, or it may be stenosed in part of its extent and dilated in another part. It may be abnormally contracted. The colon or any part of it may be rudimentary. There may be obstruction, as in the rectum, in any part of the course of the colon. There may be a congenital occlusion of the ileocæcal valve.

Finally, Jacobi has placed on record a case of congenital constipation due to misplacement of the large gut and inordinate dilatation of this viscus. In some cases of congenital malformation the small intestine may be entirely obliterated; or the small intestine in part of its extent may be normal, especially the duodenum; whereas the ileum may be rudimentary and the large gut enormously dilated. There are cases on record in which there was no connection between the large and the small intestine, and there may be congenital stricture in any part of the small intestine, either the duodenum or the ileum; or there may be an obstruction due to a small diaphragm extending into the lumen of the gut in any part of its course. It may be seen from a simple enumeration of the causes of congenital constipation that the conditions found are extremely varied, and in most cases cannot be remedied by surgical means unless the obstruction diagnosed is low down in the rectum or sigmoid flexure, and exists without any accompanying deformity of the rest of the gut. A congenital absence or rudimentary condition of the small or large gut must eventually prove fatal. The symptoms of all the cases recorded of congenital constipation are those of obstruction, in the end resulting in rejection of all fluids, vomiting, and ending fatally if unrelieved. A further discussion of this form of constipation is scarcely within the scope of this treatise.

Acquired constipation may be acute or chronic. Acute constipation is really a surgical disease, and is caused in infants and children by some acute obstruction of the gut, such as intussusception, volvulus, strangulation, through a slit in the omentum, strangulation by peritonitic bands, or by the persistence of Meckel's diverticulum; hernia of all kinds, strangulation or paralysis of the intestine as a result of traumatism. Peritonitis may cause acute constipation, and with this we must consider diseases such as appendicitis.

Foreign bodies may obstruct the lumen of the bowel. Watkins has recorded in the *Lancet* the case of a boy, ten years of age, who had swallowed an immense quantity of figs, which obstructed the lower part of the gut near the anus, and had to be removed by surgical means before movements were established. J. Lewis Smith relates the case of a girl, four years old, in whom acute constipation developed suddenly as the result of the impaction of a mass of intertwined worms in the gut. This acute obstruction was attended by distention of the abdomen and great suffering. A large gallstone is mentioned as obstructing the ileocæcal valve, and in this way suspending for a time the passage of fæces through this structure.

The diagnosis of acute constipation presupposes a diagnosis of the primary causal condition, and this can only be made by a careful study of the case. Cases of intussusception, volvulus, strangulation of the gut, either by bands or hernia or forms of peritonitis, will give symptoms of these diseases. It is scarcely the place here to enter upon these fully. In those cases in which worms obstruct the gut, the diagnosis can only be made after relief has been established by passage of the *corpus delictu*, unless enough fæces are voided to examine the same for eggs of the worms.

Chronic constipation may be dependent upon obstruction of the large or small gut in any part of its extent, either by morbid growths, sarcomata, carcinomata, or tuberculous peritonitis. The latter form of obstruction of the gut by tuberculous masses is of especial interest, inasmuch as these cases form a part of the symptomatology of tuberculous peritonitis. I saw recently a case of tuberculous peritonitis in which large masses were palpable in the abdomen, and in which one of these masses involved the descending colon to such an extent as to almost completely occlude its lumen.

Anal fissure is a common cause of chronic constipation in infants and children. In these cases there is always a history of great pain when the movement is passed, and for some time afterward. Blood may accompany movements when there is a fissure of the anus. Children suffering in this manner do not void a movement for days, and when the movement is passed the suffering sometimes is intense. In some children there is a spasm of the anus due to a nervous condition, and sometimes brought about by an excoriated state of the anus. Examination does not reveal any fissure, but there is a distinct spasm of the sphincter which prevents the successful evacuation of the rectum. In all of these cases chronic constipation is really a surgical disease, and can only be relieved by surgical measures. In some cases caused by cancerous, sarcomatous, or tuberculous growths even the surgeon is helpless to relieve the patient. Constipation caused by anal fissure, spasms of the sphincter, or excoriations around the anus yields more successfully to surgical treatment, which is the same as a treatment for similar conditions in the adult, viz., forcible dilatation of the sphincter.

Chronic Habitual Constipation.—The next form of chronic constipation which we will consider is that which most interests the general practitioner, and is known as chronic habitual constipation. Of all the conditions within the domain of pediatrics habitual constipation is the most difficult of management. It is not always possible in these infants and children to fix on the absolute causes of a constipated habit.

Breast-fed Infants.—Infants at the breast may be constipated from birth, though normal in every other respect, and continue this habit throughout childhood. In many of these cases the mother is of a constipated habit. Some signs of rachitis may be present in certain cases. In these cases, however, it is reasonable to conclude that the mother's milk is lacking in some element, such as fat, which tends to perpetuate the constipation. In other cases the milk may be absolutely normal, and still a condition of atony of the gut of an hereditary type may exist.

Constitutional Dyscrasia.—Rachitis, when marked, is associated with constipation in a large proportion of cases. In a manner similar to the bones, so the muscular apparatus lacks tone, and it is not surprising that with the muscular atony the glandular elements of the gut should be deficient in furnishing elements necessary to a normal maintenance of the functions of the gut and evacuation of the intestinal contents.

Heredity has been named as a cause of constipation in breast-fed infants, and it is not infrequent to meet the same condition, possibly due to the same cause, in bottle-fed infants. Incorrect feeding is certainly one of the most frequent causes of constipation in artificially fed infants and children. Some infants who have been started on very dilute modifications of milk are constipated from the beginning, or their constipation has been fostered by heating the milk to a greater or less degree, and in these cases the constipation, if allowed to persist for any length of time, is perpetuated into the period of childhood. In older children a simple diet of two or three articles of food, which have been religiously adhered to from the time of weaning to a varying period of childhood, is the direct cause of constipation. There has been a failure in these cases to give an appropriately mixed diet. I have seen constipated children, at varying periods of childhood, who have been kept systematically on a diet of milk and fruits, for fear that any other article of diet would cause intestinal disturbance. The result has been an inordinate constipation of chronic duration with accompanying symptoms.

Symptoms.—One can scarcely speak of the symptoms of a condition which in itself is a symptom of disturbed conditions. There are certain features, however, of the movements of constipated infants and children which are of importance.

The **movements** of infants suffering from constipation may be hard and formed, or may be unformed and dry. Ordinarily a healthy infant has two, three, or four movements daily, the rule being two. A healthy infant may have six movements a day and still be within the limits of health. We judge by the character rather than by the number of the movements. The movements of infants and their normal characteristics have been dilated upon elsewhere, and the reader is referred to the section treating of this subject.

In constipated infants the movements consist almost entirely of marble-like masses, resembling those seen in the lower animals. They rarely have a movement unaided. They have great pain in passing the movements, and in time develop fissuration of the anus to a greater or less extent, with accompanying bleeding due to the stretching of the fissure. In other cases this bleeding is accompanied by slight prolapsus of the gut during the movement, which often creates the impression that the infant is suffering from hemorrhoids. Many of these constipated movements are coated with mucus, or mucus is voided after the movement is passed. These masses are not membranous, and if examined will be seen to be composed mostly of mucus. Constipated infants after a time develop a pallor and anæmia which is characteristic, and seem to suffer from intestinal absorption and toxæmia which results from time to time in periodical attacks of vomiting, which is discussed elsewhere. These children also complain from time to time of a vertigo and nausea, especially in the morning. Many children who are thus constipated will reject their food in the morning. They lose their appetite and have all the symptoms of intestinal intoxication.

The **treatment** of constipation is dietetic and medicinal. If the infants who are constipated are fed at the mother's or nurse's breast, the bowels of the mother or nurse need regulating, and they should take regular exercise. In many cases a nutritious diet to the mother or nurse will cause the milk to change in its composition, containing more fat, and thus improve the condition in the infant. This is not always the case, however, and the most baffling cases of constipation are those which exist in infants at the breast. It has been suggested by Biedert and Holt to give such infants a small quantity of cream after each feeding; but even this procedure does not succeed in many cases, and in the summer is distinctly dangerous, inasmuch as the mixed feeding may disagree with the infant. A teaspoonful of cream containing 16 per cent. of fat, or a teaspoonful of the nine-ounce top milk taken from a quart of milk is given three or four times daily before or after nursing, and in some exceptional cases the bowels can be regulated. As has been intimated, some infants on this treatment begin to vomit and develop a diarrhœa, especially in the summer season. This diarrhœa is not necessarily an infectious one, but is rather a fat-diarrhœa, and is as difficult to control as some of the infectious varieties. It is scarcely advisable to pursue this method of treatment during the summer season. If artificially fed children are constipated, the heating of the milk should be stopped. If for some reason milk must be pasteurized or sterilized, the time of heating should be reduced to a minimum. Constipated infants may be fed on raw milk if the milk is fresh and carefully kept. The formula should contain sufficient fat to make the diet nutritious, but the fat should not form more than 4 per cent. of the mixture. As a rule, artificially fed infants do well on a smaller quantity of fat than the average breast-fed infant. Thus 2.5 to 3 per cent. of fat meet the requirements of most infants. If they are constipated the fats are raised to 4 per cent. This proportion should not be increased, since there is danger of disturbing the functions of the gut to such an extent as to give rise to conditions more serious than the constipation.

Children from the sixteenth month to the second year who suffer from constipation should be gradually weaned to a mixed diet. In many cases this procedure will regulate the bowels. The children should be given green vegetables, such as peas and spinach, in the form of a *purée*. The diet should include cereals of the various varieties, especially wheatena, oatmeal, granum, and rusk (Zwieback). The milk should be given raw with a moderate mixture of cream. Fruit, such as oranges, raw apples, and pears, is also given in moderation. If the constipation cannot be remedied by these measures, recourse is had to medicinal treatment.

Cathartics.—At best, cathartics are a makeshift. Some older children will do well with a small dose, grain $\frac{1}{150}$ (0.0004), of strychnine once a day, and a simple cathartic, such as the aromatic fluid extract of cascara, twice or three times a week. A child two years of age may be given ♏ xx to xxx (1.0 to 2.0) once a day. The preparations of rhubarb are useful, but do not give uniformly satisfactory results. The mercurial cathartics are available only once a week in the majority of cases. We are thus reduced to the necessity of giving suppositories or enemata. With very young infants a small cylindrical piece of soap inserted with oil into the rectum once a day will be effective. With older children the glycerin suppository given every other day is very useful.

Enemata.—In many cases it is necessary to give enemata : to younger infants they are given once a day ; to older children affected with the form of constipation occurring in connection with mucous colitis, an enema is given twice a week. The diet in these cases of mucous colitis should be regulated. When the child becomes pale and listless a brisk cathartic aided by a large high enema is given. In this way the attack of vomiting may be avoided.

Massage.—Massage of the abdomen gives very unsatisfactory results. Gymnastics or calisthenic exercises in the morning after a bath are useful in some cases.

Useful formulæ are the following:

1. Pulv. glycyrrhizæ comp. . .	ʒss to ʒj (2.0 to 4.0) as necessary.
2. Infus. sennæ comp.	ʒj–ʒij (4.0–8.0) as necessary.
3. Podophyllin	gr ij (0.12).
Syr. rhei arom.	℥ij (60.0).
Sig. ʒj (4.0) pro dosi.	

Congenital Dilatation of the Colon, With or Without Hypertrophy of Its Walls.

This deformity is one of the rarer causes of habitual constipation in infants and children. We distinguish three forms of this condition:

a. In this form there is an increase in the length of the colon descendens and the sigmoid flexure. As a result of the increased length of the colon this portion of the intestine bends two or three times on itself. There is a stagnation of the fæces and consequent constipation. Toxæmia results and emaciation follows. With the above there are symptoms of fermentation in the gut, and constipation alternates with diarrhœa. The diarrhœal movements are foul, containing mucus and blood. There is some meteorism.

The **prognosis** of this form is not bad, provided a complicating colitis does not ensue. As the child grows older the above symptoms improve and normal conditions ultimately supervene.

b. In this class of cases the colon is not only lengthened and dilated, but its walls are thickened. Such are the cases of Mya, Formad, Griffith, and Hirschsprung. According to Concetti, the mucosa is not only thickened, but the connective tissue and muscular coats of the intestine show the same changes, and the arteries are the seat of arteritis. The cases belonging to this class in the literature range from eight to fifty years of age. It is in this set of cases that stagnation of the fæces is accompanied at times with ulceration of the gut.

c. In this class of cases there is a combination of the dilatation of the colon with thin walls; or the colon may be normal in its lower portion and slightly ectatic, with hypertrophied walls above.

The **symptoms** of the last two sets of cases are more severe in the younger and milder in the older children. They are severe if the condition has lasted for two or three years, and milder if the patient has survived until the tenth or twelfth year. From the second to the fourth day after birth great meteorism appears. No meconium is passed for some time, and there is no stenosis of the gut; laxatives succeed in bringing away only a small amount of meconium or fæces. The constipation is very obstinate, the fæces are foul-smelling, and from time to time colitis may supervene, or every eight to thirty days hard, malodorous masses are evacuated with slime and blood. There is a condition of an auto-intoxication and a resultant cachexia. The abdomen becomes enormously distended, and the coils of the gut can be made out on the surface. The children die during the first and second years of life, either through cachexia or perforation of the gut. Of the 21 cases collected by Concetti only 2 lived. One was a case of his own, and another that of Osler; in both an artificial anus was made for the relief of the condition. Colitis, with or without perforation of the gut, is the most frequent cause of death. The remaining cases die of cachexia.

Treatment.—The first class of cases are treated in much the same manner as is constipation. In the second and third forms surgical interference is indicated as soon as the diagnosis is made. The colon is resected. Thus far surgical interference has not been attended with great success.

Acute Intestinal Obstruction.

(*Intussusception.*)

Intussusception, according to Treves, is the prolapse of one part of the intestine into the lumen of an immediately adjoining part. It causes more than one-third of all the varieties of obstruction of the gut.

Varieties.—Invagination of the gut may take place in any part, from the duodenum to the rectum. There are the following forms:

The **enteric form,** which may involve any part of the small intestine, but which most commonly involves the lower part of the jejunum or the ileum.

The **colic form,** which may involve any portion of the colon.

The **ileocæcal,** which is the most common form.

In the ileocæcal variety the ileum and cæcum pass into the colon, the valve preceding and forming the apex of the intussusception. In the ileocolic form, the valve remains stationary and the ileum passes into the colon. In the latter form there is an invagination of the cæcum and colon, of a secondary character.

Etiology.—Nothnagel demonstrated that intussusception is caused by irregular muscular action in the wall of the gut; in acute intussusception this is of a spasmodic character. In 50 per cent. of the cases little is known of the exciting cause.

Diarrhœa, the various forms of enteritis, polypi, and diverticula, improper food, traumatism, and exposure to cold, have all been regarded as exciting causes. Typhoid fever and pertussis have been complicated or followed by intussusception. I have recently seen a case following typhoid fever in a boy three years old.

Meckel's diverticulum and the appendix have been the cause and seat of intussusception. In the latter case the inverted appendix caused ileocæcal intussuception.

Frequency.—Intussusception is more common in males than in females. The disproportion diminishes after the first year of life. Fifty per cent. of all the cases occur before the tenth year, and chiefly in individuals who are not in good physical condition (Treves). In the cases that I have seen, the infants were delicate, the child being robust in only one case.

The youngest case I have met was five and a half months of age. This infant was breast fed, had suffered with colic, and had had green movements from birth; there was an ileocæcal invagination eight inches in length.

Symptoms.—The onset is sudden in 75 per cent. of the cases; in the colic and rectal varieties it may be gradual. In many cases the disease makes its appearance while the infant is nursing or during sleep. The patient, being attacked with pain, suddenly awakes from sleep with a cry and begins to vomit; on the same day or the following day a bloody movement appears, the amount of feces being small. In a few cases there are no fecal evacuations. If the case is progressive, the pain returns in paroxysms, the hemorrhagic movements are repeated, and the vomiting keeps pace with the increase of the obstruction. The general condition of the patient grows worse; apathy and collapse ensue. I have seen cases begin with a mild diarrhœa; the pain suddenly appears, and also the hemorrhages from the bowel, the infant at once going into collapse. There is apathy, from which it is difficult to rouse the patient. If the case continues to progress, the movements become frequent, exhaustion increases, and finally death from asthenia results. The pain is great at the onset, usually reaches its maximum intensity within a short time, and then gradually subsides. It is of a paroxysmal character and is colicky during the advance of the invagination; as adhesion takes place or gangrene occurs it diminishes. The intervals between the paroxysms of pain are at first of considerable length; later they become shorter. The pain is most severe in the ileocæcal form, and is in all forms caused by irregular intestinal peristalsis.

Vomiting is not so prominent a symptom as in other forms of intestinal obstruction (Treves). In 75 per cent. of the cases it comes on early with or directly after the pain. It may not recur for hours. In a child taken with sudden pain of a colicky character, vomiting, and bloody stools, the vomiting recurred only twice within twenty-four hours. It is apt to be less violent as long as there is not complete obstruction of the gut; in other words, it is more marked in those cases in which no feces pass through the gut. As long as the pain recurs in paroxysms (progression of the intussusceptum) the vomiting is not apt to be marked. The vomited matter is composed of the stomach contents and is biliary; stercoraceous vomiting was found late in only 25 per cent. of Leichtenstern's cases; Gibson also found it to be rare and late. If stercoraceous vomiting was present, it appeared from the fourth to the seventh or to the fourteenth day. In the case referred to, in the infant of five and one-half months, it appeared during the first twelve hours of the disease.

The condition of the bowel is important. It is generally stated that constipation occurs from the outset; this is not universally true. Cases in which constipation exists throughout, that is to say, in which no feces whatever are passed, are not common, and form only 30 per cent. of the total number. Diarrhœa is the common condition at the outset; as the obstruction increases, the amount of feces in the stools diminishes, and finally only mucus and blood are passed.

The most important symptom in connection with the bowels is hemorrhage. Hemorrhage from the bowel, in connection with pain and other abdominal symptoms, is considered by Gibson as pathognomonic. It was present in 80 per cent. of the cases tabulated by Leichtenstern. As a rule it is considerable. It is said by Treves to have been in some cases so great as to cause death. The blood and feces have a cadaveric odor, which however is not always, as some writers affirm, a sign of gangrene. I have perceived this odor in an intussusception which operation showed not to be the seat of gangrene. It is caused by decomposition of the blood in the gut.

The temperature is normal, slightly subnormal, or slightly elevated. There may be a slight elevation of temperature without peritonitis. The quantity of urine may as in other forms of intestinal obstruction be diminished.

Tenesmus is present in 55 per cent. of the cases; it depends more or less on the presence of the intussusception in the rectum. It is usually an early symptom in the rectal form, and is more common in the ileocæcal variety than in the enteric.

The abdomen is not at first distended; it may, on the contrary, be retracted; if tympanites occurs at all, it does so late and in the presence of a general peritonitis. Palpation of the abdomen is at first well borne, but after a time there is sensitiveness.

A **tumor** felt through the abdominal wall or in the rectum is of the greatest value in the diagnosis. It cannot be felt if the intussusception is in the hepatic or splenic flexure of the colon. It is variable in distinctness, and is most frequently felt in the region of the descending colon or of the sigmoid flexure. It is hard and resistant, and rarely more than six inches long. It is often said to be sausage-shaped, but the statement is misleading. The tumor is rarely felt in the ileocæcal region, for the reason that the intussusception in this locality is small, and is that of a small gut inside of a large one. In one-third of the cases the rectum, if examined, shows the presence of the intussusceptum. The rectal tumor is commonly found in children, because in them the colon is mobile. The gut may reach the anus as early as the second day, the average time being the seventh day. It may protrude from the anus from three to eight inches, and may be in a gangrenous state; under these conditions it has been mistaken for a polypus or hemorrhoid.

Prognosis.—As regards duration, there are three varieties of intussusception—the ultra acute, the acute, and the subacute. The ultra acute cases are exceedingly rare. Leichtenstern found only 5 of this form in a total of 7269 cases; 4 of the 5 occurred in infants less than a year old. All were fatal.

The rate of mortality in intussusception, excluding the ultra acute forms, varies as given in the statements of different authors. Gibson's statistics place the mortality at 53 per cent. It varies with the age of the patient, the duration of the disease before operating, and the success in reducing the intussusception. Intussusception is extremely fatal in infants under the first year. Thus according to Treves, the mortality under one year of age is 80 per cent. On the other hand, if we study the cases as Gibson has done, we find that the cases operated on during the first day of the disease had a mortality of 41 per cent.; those on the fourth day, 72 per cent. The reducible cases showed a mortality of 38 per cent.; the irreducible, of 88 per cent.

Diagnosis.—From the studies made by Gibson, it may be seen that, in children, a bloody discharge with abdominal pain of a paroxysmal nature is almost pathognomonic of intussusception. The presence of a tumor fixes the diagnosis absolutely. Fecal vomiting is of very little value as a diagnostic sign. It is very infrequent, and is in any case present only late in the disease, when occlusion of the gut has occurred. If enteritis exists in a young infant, it is often difficult in the absence of any abdominal or rectal tumor to make a diagnosis. The course of the case will guide the physician. In dysentery the hemorrhage from the bowel is not great; it is composed of blood-tinged mucus. Cases of scurvy may simulate intussusception if bloody discharges appear with the intestinal movements. In these cases the amount of blood voided per rectum is fully as great as in cases of intussusception. In scurvy, however, there is fæcal matter in the movements, in the cases coming under observation of the author, as also signs of scurvy, such as tenderness of the bones and spongy, bleeding gums. Appendicitis has been mistaken for intussusception. It frequently occurs with it, and thus obscures the picture. Peritonitis can hardly be mistaken for intussusception. In peritonitis the pain is continuous and there is tympanites, but no bloody discharge. Tuberculous peritonitis is sometimes mistaken for intussusception. In tuberculous peritonitis the symptoms are not progressive, and also there is not likely to be a bloody discharge.

The case following typhoid fever, to which I referred, simulated a hemorrhage from a typhoidal ulcer. A careful examination under an anæsthetic cleared up the case. In complete relaxation under anæsthesia, a tumor could be felt in the cæcal region of the ascending colon. The result of examination was verified by operation. In all doubtful cases in which the restlessness of the child interferes with a careful examination an anæsthetic should be given. There is a characteristic condition which in some cases can be detected by examination. As the finger is inserted into the anus the rectum is felt to be inflated. This is due to traction on the gut by the invagination. I have found this inflated state of the rectum in two infants suffering from intussusception.

Spontaneous Cure.—There is little doubt of the possibility of spontaneous recovery in invagination ; such cases have been recorded by competent observers. Henoch has seen typical intussusception retrograde and the patient recover. There is another mode of recovery which occurs in cases of irreducible intussusception : the intussusceptum sloughs off and is passed per anum. This occurred in 43 per cent. of the unrelieved cases (Leichtenstern), but in 40 per cent. of these the patient succumbed to general sepsis with or without peritonitis or to subsequent obstruction of the gut from swelling after the gangrenous portion was passed. Henoch reported a case of this kind.

Treatment.—The diagnosis of intussusception once made, the case is one for surgical interference. The sooner surgical treatment is begun, the better the chances of recovery. Injections of air, gas under pressure, and enemata of water and oil have been tried, with some measure of success. Their use, however, delays the radical treatment, and apparent improvement frequently gives way to an exacerbation of symptoms. Surgical aid then comes too late. The objections to the treatment by injection are as follows : the gut is viable in these cases, and is liable to be ruptured by injection of gas or air under pressure ; an enema of water under only four feet of pressure has been known to produce this result. Snow published a case in which an injection of oil was made ; post mortem the oil was found above the point of obstruction. The enema may thus pass through the lumen of the gut without relieving the intussusception. Enemata should be given, if at all, during the first forty-eight hours, and should be allowed to flow into the rectum under very low pressure. The amount of fluid varies ; certainly not more than a quart should be given. The fluid, a saline solution at 100° F. (37.7° C.), is allowed to remain in the rectum for ten minutes, the patient being under an anæsthetic. A Davidson syringe should not be used. The ordinary bag irrigator is best for this purpose. If one enema fails and the diagnosis is certain, there should be no delay in seeking surgical assistance.

Appendicitis.

(*Perityphlitis; Paratyphlitis.*)

Anatomical Peculiarities.—Vallée examined the appendix in 100 infants and children post mortem. He found that in fully 75 per cent. the cæcum is situated above the anterior superior spine, on the right side, a position higher than that occupied in the adult. It is above the plane of the anterior superior spine of the ileum, is almost 5 centimetres long, and has a general longitudinal ascending or descending direction. In one case the appendix was situated entirely to the left of the median line, there being no transposition of the other viscera. Knowledge of these facts is of importance in the examination for the appendix in conditions of disease. I have frequently succeeded in palpating the normal appendix at one side of the cæcum. It is felt as a cylindrical body having the diameter of a quill.

Acute Appendicitis.

Frequency.—Although the statistics showing the frequency of appendicitis in infancy and childhood vary with the number of cases collected by each author, the combined statistics of Matterstock, Fitz, Sonnenburg, and Nothnagel, show that the disease is not very frequent before the tenth year. Only 8 per cent. of the cases occur at this age. It may occur in early infancy. Savage records a case in an infant two months old ; Demme also records a case in a very young infant.

The literature shows occasional cases at all periods of infancy. Among the cases collected and tabulated from the service of my colleagues, Gerster and Lillienthal, at the Mount Sinai Hospital, there is one of an infant one year of age. Of 50 cases of appendicitis in children taken from the service of these surgeons, 1 occurred in an infant one year of age, 17 from the third to the sixth year, and 32 from the sixth to the tenth year of life. Thus in a statistical collection of cases occurring in children, only one-third occurred before the sixth year of life.

The **forms of the disease** are the same as in the adult subject. The perforative form seems to be the most common among children. Thus of 50 cases coming to the hospital for operation, 31 were perforative with or without abscess, 9 were of the gangrenous variety, and 6 of the catarrhal form. It will thus be seen that in children the tendency in this disease as in others, such as pleurisy, is toward suppuration and the formation of abscess.

The **symptoms** will vary with the variety, whether catarrhal, perforative, or gangrenous.

a. In the **catarrhal form** the patient is, after some indiscretion in diet, seized with colicky abdominal pain, vomiting, and some fever. In other cases the children simply complain of pain which is not sufficiently severe to prevent their being up and about. The pain is not always located by the patient in the appendix. When the patients are in the recumbent posture, the right knee may be flexed and the thigh flexed on the abdomen; when they walk, they do so in a bent position, favoring the affected side. Physical examination reveals a localized resistance or tenderness in the right iliac fossa. In some cases there is distention of the cæcum with feces, in others I have felt the appendix and the cæcum matted together in a mass of the size of the index finger.

The pain is not always referred to the iliac fossa, but may be distinctly located around the umbilicus or over the lower part of the abdomen.

It may not always be possible to palpate the appendix, which may be behind the cæcum. Under such conditions no intumescence will be found. McBurney's point will be considered in the diagnosis.

The history of many of the catarrhal cases is one of recovery under careful treatment. The fever subsides or may never have been above 101° F. (38.3° C.); the pain also subsides, and in from a few days to a week the patient is apparently well. Attacks of this kind may recur.

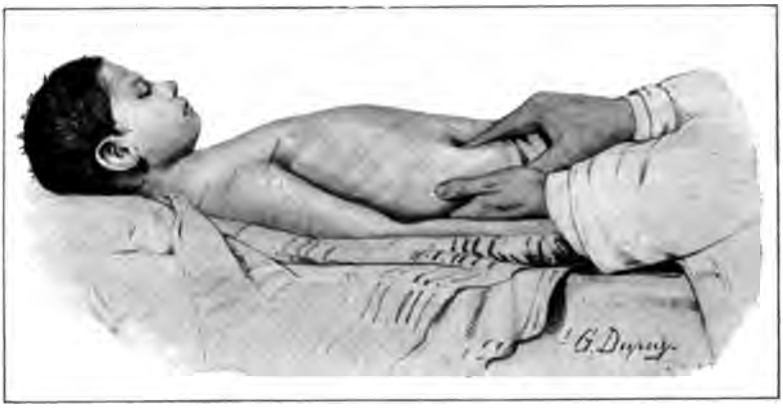
b. In the **perforative** or **suppurative form** the symptoms are more violent. In this form also the onset of the disease seems to date from some indiscretion in diet. The patient is seized with sudden sharp pains in the abdomen, accompanied by vomiting, fever, and rapidity of pulse. The pain is located either in the upper or the lower part of the abdomen, or in a few cases in the right iliac fossa. After one or two attacks of vomiting this symptom may subside and not recur until the second or third day, when perforation occurs. Tympanites occurs early and may set in after the second day of the disease. The pain and tympanites cause an increase in the respiratory movements, which are shallow. The patients lie in the recumbent posture. The tympanites, if the perforation is extensive and there is general peritonitis, causes, as in all forms of perforation, a disappearance of the liver dulness. The pulse is at first rapid and thready, and quickly mounts above 120 after perforation has occurred. The prostration is great, and in some cases of a septic type jaundice is present.

c. In the **gangrenous form** the symptoms are very similar to those of the perforative form, but are very much intensified. It is not possible to tell from the symptoms whether the process is gangrenous, simply perforative, or catarrhal followed by abscess.

Course.—In both the perforative and the gangrenous cases in children as in the adult, localized adhesions may form with a small or large collection of pus or several foci of pus. In other cases a general peritonitis follows the perforation. In children, as in adults, the moment of perforation is followed by a temporary fall in the temperature and a cessation in the pain and vomiting, the pulse, however, continuing rapid. The lull, however, is of short duration, and is quickly followed by an increase in the severity of the symptoms.

Diagnosis.—The above outline gives very little idea of the great and sometimes insurmountable difficulties of diagnosis of appendicitis in young children. To guard against error, a very careful routine should be followed. The patient is completely undressed and lies in the recumbent posture, the shoulders being slightly raised. The physician should stand or sit at the patient's right. The contour of the abdomen is noted. If it is normal and not distended, there is probably no peritonitis. The abdomen is very gently palpated in different places to ascertain if there is distributed or localized tenderness.

FIG. 99.



Method of examination of the region of the appendix vermiformis.

The left palm is then placed underneath the right loin, and with the palmar surface of the fingers of the right hand the region of the appendix is gently palpated (Fig. 99). Superficial palpation is practised at first. The hand is then depressed deeper into the iliac fossa in search of resistance or tumor. The intensity of the pain caused by manipulation is carefully gauged by watching the face of the patient. The right iliac region having being carefully palpated, rectal exploration should be made in all doubtful cases. This is necessary in the cases in which a general tympanites or general abdominal tenderness makes the diagnosis difficult. With the well-oiled index finger of the right hand the rectum is explored as high up as possible. In young children this can be done without causing pain if gentleness and caution are exercised. If children are very intractable, this method of examination cannot be carried out.

The following points are important in the diagnosis:

Tympanites.—If the abdomen is distended and there is general pain with increase of the number of respirations, there is probably peritonitis localized or diffuse. In the latter case there is disappearance of the liver dulness if the tympanites is extreme.

Percussion will sometimes, even in general peritonitis, give a localized dulness in the right iliac fossa. Localized pain and intumescence or a localized mass in the right iliac fossa are of great import.

McBurney's point is of less value in children than in the adult. In children, as will be seen from Valleé's work, the appendix is situated higher than in the adult, and McBurney's point is therefore too low for palpation. Some children complain of epigastric, others of umbilical pain, which is not so distinctly localized as in the adult.

The **fever** is of little value, there being nothing characteristic in the curve. The temperature may be normal or in severely septic cases slightly raised. After perforation, the temperature becomes subnormal, as it does in the adult.

Appendicitis in children may simulate **tuberculous peritonitis.** In the latter disease there is sometimes severe pain of the colicky variety. Tuberculous peritonitis and appendicitis may be coincident.

Pain in appendicitis resembles very closely that in gastro-enteritis and dysentery. Griffith has published 2 cases of appendicitis in children who had entero-colitis at the same time.

I have had one case in which a **perinephritic abscess** simulated an appendicitis. The contrary may also occur. Appendicular abscess may simulate a coxalgia with abscess. I have seen a few cases of typhoidal affection of the appendix which for a few days simulated an appendicitis very closely. Appendicitis with invagination of the appendix into the cæcum is a rare condition, as is also intussusception with appendicitis. In the typhoidal cases, a Widal reaction may be obtained, and will be of assistance in diagnosis. Care should be taken that a perforating typhoidal ulcer does not escape diagnosis. Intussusception will give the characteristic symptoms of that condition.

I have seen cases of **lobar pneumonia** of the lower lobe of the right lung, in which the pleuritic pain radiated down the right side into the iliac fossa. There were also epigastric pain and vomiting at the onset of the disease. The excessive rapidity of the respirations, the marked dyspnœa, and absence of tympanites and pain on deep pressure in the region of the appendix, led me to examine the lung.

Prognosis.—Of the 50 hospital cases which I have tabulated above, only 3 recovered without operation; they were of the catarrhal variety. These figures give no accurate idea of the proportion of recoveries made under careful and conservative treatment in private practice.

The mortality in the cases operated upon was 35 per cent. The rate is not high considering that many cases came under the knife later than would have been the case in private practice. On the other hand, it should be remembered that the rate of mortality is also influenced by the nature of the infection and the power of resistance of the patient. Thus cases with a gangrenous appendix died although operated upon on the second day; others of the same kind recovered although the disease had lasted from four to seven days before operation. Some perforative cases died on the second or third day of the disease, while others recovered although operated upon from six to twelve days after the onset of symptoms. Gangrenous cases in this statistical table in children show a lower rate of mortality than those cases in which the appendix perforates, forms an abscess, and causes general peritonitis.

Chronic Appendicitis.

This form of appendicitis occurs in older children. The cases are frequently mistaken for those of dyspepsia, and *vice versa*. The history is much the same as in the adult. A child otherwise in good health has attacks during which there is abdominal pain not of great severity, but which may last for a few hours and disappear, leaving the patient well. The pain is very rarely referred to the appendix; it is abdominal, the umbilical region being generally indicated as the seat of discomfort. The temperature may reach 100° F. (37.7° C.); the pulse in a child of eight years was 96 and regular. There is no vomiting and no prostration. The pain is sufficiently severe to make the patient wish to lie down; it is not excessive when the appendix is palpated. The bowels are regular. The cases may in the intervals between the attacks show a slight intumescence in the region of the appendix, but nothing is felt in the rectum. The signs in the interval may be very indefinite or quite distinct. The cæcum and appendix are felt to be matted together. Three cases in which there had been repeated attacks extending over a period of from one to two years, were operated upon for me by leading surgeons. The patients were girls between the ages of six and eight years. In each case there was evidence of a chronic catarrhal process. In one case the appendix contained a fecal calculus, in another there were constricting adhesions.

The **treatment** of both acute and chronic appendicitis in infants and children does not differ from that followed in the adult subject.

Rectum.

In infants a large portion of the rectum is situated in the abdominal cavity rather than in the pelvis. It has three curves—one lateral and two anteroposterior. The gut is nearly straight and occupies a more or less vertical position, hence the frequency of prolapse. The attachment of the rectum to the surrounding parts is not extended as high in children as in adults, hence the rectum is more liable to be pushed out. The rectum of the newborn infant may be divided into three parts. The first lies in front of the sacrum and ends at the lower end of the bone; the second is short, and in this respect differs from the adult gut, being also more vertical; the third portion is long, and extends downward and somewhat backward. The second portion being short, when the rectum is distended, the gut is straightened out and the whole rectum extends downward and backward (Symington). All these data are of importance in applying methods of therapy (enteroclysis, etc.) to this organ.

Prolapsus Ani.

Prolapsus ani is a condition frequently met with in infants and children. It may amount only to an eversion of the mucous membrane. There is in some cases a complete descent of part of the rectum, which protrudes from the anus to the length of one or two inches.

The **etiology** of this condition is obscure. It evidently occurs only in cases in which the pelvic attachments of the lower bowel are lax. It is favored by anatomical conditions elsewhere mentioned. It is seen in children who are constipated, in those who suffer from diarrhœa, and also in those whose movements are not normal. Any abnormal condition in the neighboring organs, such as the bladder and urethra (stone), may cause excessive straining and consequent prolapse of the gut. A rectal polypus may cause prolapse.

Symptoms.—In some cases the only symptom is the appearance of a small quantity of mucus and blood on the diaper with each movement; in these cases the prolapse returns spontaneously. In other cases the bowel descends to the extent of one or two inches with the movement, and remains prolapsed. If a polypus of the lower part of the rectum is the cause of the prolapse, it is seen protruding from the prolapsed portion.

Treatment.—The first step is to replace the protruding gut. The gut is anointed with olive oil or vaseline and gently replaced with a towel. The movements are so regulated by diet and cathartics that the stools are passed without straining. Three times daily a suppository containing grains ij to iij (0.12 to 0.2) of tannic acid is placed in the lower bowel. While the movements are being passed the patient is kept in the recumbent posture on a bedpan. This treatment is frequently successful. In other cases, the buttocks are drawn together by adhesive straps and the child is allowed to pass movements thus strapped. Cocaine and strychnine are used both in suppositories and hypodermically. The protruding portion is painted with cocaine. These measures have their failures and successes. The only satisfactory method is that first advised—of a strict diet, the recumbent posture at stool, and the astringent suppository. The Paquelin cautery is sometimes employed to cauterize the mucous membrane. The danger in this method is the substitution of a traumatic stricture of the anus for the comparatively harmless prolapse. Application of the pure stick of silver nitrate to the anus twice a week, has given good results. If a polypus of the rectum is the cause of the prolapse, the growth should be removed by surgical means.

Fissure of the Anus.

Fissure of the anus is seen in syphilitic infants, in those suffering from marked constipation, and in infants that have eczema of the anus. It may be the result of the repeated introduction of the hard nozzle of an enema syringe. The fissure may be so slight as to be only a line-like tearing of the mucous membrane, or may consist of a broad ulcer with a hard granulating base.

Symptoms.—As a rule, the infants are constipated. When a movement is passed, the infant cries and there is great pain. A few drops of blood are passed on the diaper.

Diagnosis.—The presence of a fissure of the anus sometimes escapes the notice of the physician. If there is a history of the above symptoms, the physician should place the infant on a table, grasp the buttocks with the palm of the hands and separate them forcibly with the thumb. The anus is thus everted, and if a fissure is present it will at once become apparent.

Treatment.—A small fissure is sometimes very successfully treated by regulating the bowels. It is touched with a 10 per cent. solution of silver nitrate once a day. In the severe cases silver applications will not avail; forcible dilatation of the rectum by means of the thumbs must be resorted to. This procedure not only cures the fissure, but is also an effectual remedy for the accompanying constipation.

Spasm of the Anus.

Cases of nervous spasm of the sphincter ani occur in infants. The infant is constipated and cries at each movement. There is no bleeding, nor does examination reveal any fissure, but only marked contracture of the anal opening. In these cases it is almost impossible in an examination to bring down the upper part of the anal gut.

The remedy is to regulate the bowels. If by this means success in overcoming the spasm is not attained, forcible dilatation is the only resource.

Proctitis.

Apart from the membranous and catarrhal forms of proctitis, which occur with similar conditions of the intestine, the only form which is of interest is the gonorrhœal. This occurs as a complication of vulvovaginal gonorrhœal inflammation. In these cases the introduction of the gonococcus from the vagina into the gut has occurred through careless thermometry or the giving of enemata without previous cleansing of the parts. The disease is very painful and at the same time trying to the infant or child. With the discharge of pus from the anus there are tenesmus and a bloody discharge with the movements. The purulent discharge shows gonococci.

The **treatment** consists in the injection of protargol solutions, 2 per cent., at a temperature of 105° to 108° F. (40.5° to 42.5° C.), into the rectum twice daily. The bowels are regulated. Suppositories of tannin or tannigen are also of value and give great relief; one containing grains iij (0.18) is given per rectum twice daily. In the later stages it may be necessary to paint the lower bowel with a very weak solution (0.5 per cent.) of silver nitrate.

Polypus of the Rectum.

Polypus of the rectum is not rare in childhood, but is not often seen in infancy. It occurs most frequently from the third to the seventh year of life. The polypi are adenomata. I have examined several, and have found them to have the structure described by Baginsky. They may be single or multiple, usually have a pedicle, but may be attached to the wall of the gut by a broad base. As a rule they are situated on the posterior wall of the rectum seven or eight centimetres above the anal ring, but may be on the anterior wall. In most cases the polypi exist here only, but I have seen them higher up in the gut, and in one case in a child of five years from whom several rectal polypi had previously been removed, I diagnosed a number in the descending colon. In this case laparotomy and incision of the gut proved the diagnosis to have been correct. The polypi may, if they become numerous, assume a malignant character; this is especially true of the growths with a large, broad intestinal base.

Symptoms.—The characteristic symptom is intermittent hemorrhages from the gut, which may be profuse. At times the outer surface of the movements is streaked with blood, the bowels being constipated or normal, with an occasional mucous diarrhœa. If the polypus is low down, there is straining at stool with prolapsus of the gut. Many of the children thus affected are pale, have a pasty hue of the skin, and show evidences of lymphatism.

Diagnosis.—Bleeding from the bowel, in the absence of other symptoms, should at once suggest the necessity of digital exploration of the lower bowel. If a polypus is not found, a careful palpation of the abdomen made when the patient is fasting should be the next procedure. If the child is tractable and the abdomen soft, it may be possible to feel a tumor of the size of a hazelnut at one side of the umbilicus.

The **prognosis** is good; removal of the polypi is rarely followed by recurrence of symptoms, even in cases in which they are situated in the descending colon. If they are removable and not very numerous, the patient recovers.

Treatment.—If the polypus is low down and pedunculated, it may easily be snared with or without the aid of a rectal speculum, and crushed or ligated off. If it is high in the sigmoid flexure, the anus should be dilated and the growth reached by means of a speculum. In cases in which the growth is in the colon, laparotomy, enterotomy, and ligation are indicated.

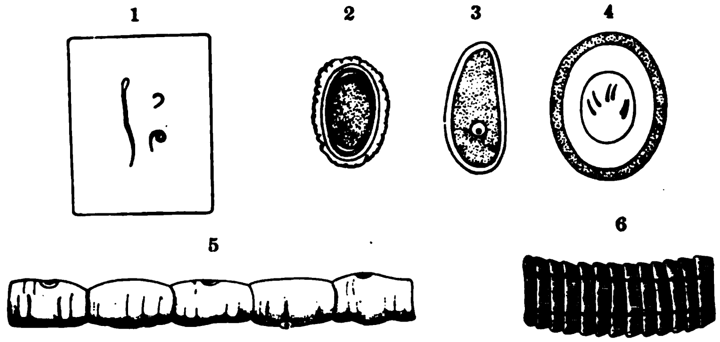
INTESTINAL PARASITES.

The most common parasites found in infants and children are the Nematoda, or round worms, and the Cestoda, or tapeworms. The round worm is smooth and light brown or reddish in color, the female being larger than the male. The eggs are found in the stools; they are from 0.05 to 0.06 mm. in diameter and are surrounded by an albuminous envelope. The worm is several inches long. Oxyuris vermicularis is about 1 cm. long, the male having a length of 4 mm. The eggs measure 0.05 mm. in their long diameter.

The tapeworms in mature state consist of rectangular segments. The head and neck are called the scolex; the segments, proglottides. The worms are hermaphrodites. The solium is sometimes several metres long. The head is of the size of a pin's head, with a projecting proboscis armed with hooklets. The eggs of the solium are ovoidal, 0.3 mm. in diameter. The Tænia mediocanellata has a more cuboidal head without hooklets (Fig. 100).

Diagnosis.—There are no symptoms which can be traced to the presence of these worms in the gut. If they increase in enormous numbers, they may cause symptoms of mechanical obstruction. Without the presence of the eggs or links of the worm, a diagnosis is not possible. Their presence is made known by the passage per anus of the links of such worms as the tapeworm. Round worms may also pass out of the anus, or may be vomited if they gain access to the stomach. Thread worms may cause excessive pruritus, and may not be discovered external to the anus. In that case the feces should be carefully examined for the eggs of the worms.

FIG. 100.



1. Oxyuris vermicularis, pin worm, natural size.
2. Egg of Ascaris lumbricoides.
3. Egg of Oxyuris vermicularis, pin worm.
4. Egg of Tænia solium.
5. Proglottides or links of Tænia solium.
6. Proglottides of Bothriocephalus latus.

Round Worms.

(*Ascarides Lumbricoides.*)

This parasite is found in the small gut; it may invade the stomach or may pass downward into the rectum. Cases are recorded (Borger) in which it has passed into the bile-duct and caused abscess of the liver. There may be only one or many of these worms in the gut. Leuckart states that they may form large masses in the gut, and thus cause intestinal obstruction. They have been known to perforate the gut and cause peritonitis. The eggs are introduced into the gut through the medium of drinking-water, fruit, and vegetables. Epstein cultivated the eggs outside of the body and then introduced them into the gut, where they developed. The male worm is 250 mm. long, the female being longer.

The **symptoms** caused when these parasites have once gained access to the body are not characteristic. I have seen the worms passed or vomited by children apparently in normal condition.

The **treatment** consists in placing the patient on a milk diet. After a few days the following powder is administered two or three times daily:

Calomel,
Santonin . . . . āā gr. ¼ (0.016).

Santonin is sometimes administered in the form of pastiles, but is not more satisfactory than the above preparation,

Oxyuris Vermicularis.

(*Pin Worm; Thread Worm.*)

Brass showed that the habitat of these worms is the small intestine, whence they pass into the cæcum. The female worm lays its ova in the folds of the gut. They may pass into the stomach and thence into the mouth, but more frequently pass out of the anus into the vagina or into the prepuce and urethra. They exist in enormous numbers in the gut, are exceedingly small, and have the appearance of fibres of cotton fabric. They can be seen by spreading the nates apart. They are then found in the anus, or in female children in the fourchette. The principal symptom is intolerable pruritus, so intense as to deprive the children of sleep. This worm is found only in the human subject. It is conveyed from person to person through uncleanliness. The larvæ adhere to the fingers, and thence are introduced into food-stuffs.

Treatment.—It is a very difficult task to dislodge these worms; injections by the rectum cannot reach those higher in the intestine. The plan which I have followed, and which gives relief, is to give daily enemata of quassia wood before bedtime:

Quassia wood (ground)	℥j (31.0).
Aquæ dest.	Oj (500.0).

Make an infusion and strain.

I have in addition utilized the prescription of santonin and calomel given above for the round worms.

Schmitz recommends the administration of naphthalin, grains j to iij (0.06 to 0.18), t. i. d., for a week, after which it is discontinued for a few days, and then given again.

Tapeworm.

(*Tænia.*)

Tænia are quite common in children, and have been found in the newborn infant (Müller and Armor). Numerous cases have been recorded of the presence of these worms in infants from the third to the twelfth month. They are most frequently found between the first and the third year. The varieties most commonly found in children are: Tænia solium, Tænia mediocanellata, Tænia elliptica, Bothriocephalus latus.

Sources of Origin.—**Tænia Elliptica.**—The lice of the house-dog and cat are introduced by the fingers of the children into their mouths, and thus gain access to the gut. There the larvæ of the tapeworm which they contain develop.

Tænia Solium.—The larvæ of this worm are found in badly cooked pork or beef.

Tænia Mediocanellata.—The larvæ of this worm are found in beef. Bothriocephalus latus is introduced by the ingestion of infected fish-food.

The larvæ of tapeworm may exist in the flesh of the hare, pigeon, pheasant, chicken, goose, or duck. Ice if made from infected water may be a means of introducing the larvæ in the body. It is thus not necessarily the meat-eating children who run the danger of swallowing the larvæ of tapeworm; milk if diluted with infected water may contain them.

Symptoms.—Tapeworms may exist for months or years in the body of a child without causing untoward symptoms. As many as three varieties of the worm have been found in the same child. The symptoms are not characteristic. The passage in the movements of the links of the tænia is the only positive evidence of their presence.

Treatment.—The only successful treatment for the expulsion of the tapeworm is that which consists in the administration of filix mas in some form. It should be freshly prepared and given in liberal doses: Ext. æth. filix mas, ♏xxx (2.0) to ʒj or ʒij (4.0 or 8.0), is made into an emulsion with gum tragacanth, and mixed with equal parts of castor oil. The administration of this mixture is preceded by a day or more of milk diet. The child is then given from half a drachm to a drachm (2.0 to 4.0) of the filix mas with castor oil in divided doses. The recumbent posture is maintained in case nausea should be experienced. The movements containing the worm are carefully washed through a sieve, and the smallest part of the worm sought for in order to see if the head has come away.

The patient should be given a drawing of the comparative size of the head and links of the worm, in order that the head may not be lost, or the physician may seek it himself.

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VI. DISEASES OF THE LIVER.

Anatomical.—The weight of the liver in infants and children is from one-twentieth to one-thirtieth of the body weight; in the adult it is one-fortieth.

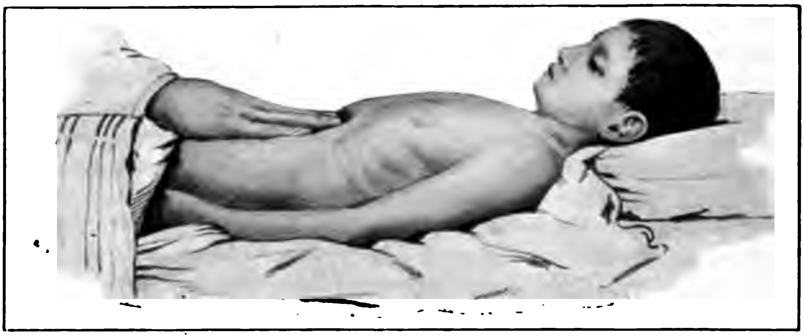
Examination.—The liver is examined with the patient in the recumbent or semirecumbent posture. The physician may palpate for the liver or mark out the organ more accurately by percussion. In marking out the organ, the upper limit, the lower edge, and the area of superficial dulness are determined. Perfect accuracy by deep percussion is not feasible, because in order to obtain absolute dulness some force must be used, and vibratory echoes of other neighboring organs—the lungs and intestines—are thus caused. In all cases it is well to determine the upper limit of dulness at a point where the liver comes in contact with the chest-wall.

The lower border of the liver is determined by palpation and percussion. The lower border projects normally in infants and children below the border of the ribs. In the right mammillary line this projection may vary from 1 to 2.5 cm. At the xiphoid appendix the liver may project to the extent of 2 to 6 cm. and still be within the normal limits. These conditions may exist up to the tenth year. The exact age at which the liver assumes the adult dimensions has not been determined. In some adults, however, the projection below the border of the ribs is the same as in children. Since the size of the liver varies, caution should be exercised in pronouncing the organ enlarged. The gut, ascites, and tympanitic distention may obscure the lower limit of the liver both to palpation and percussion.

Palpation.—By palpation, the location of the lower border of the liver may be determined, and whether it is rounded or sharp, also, if the liver be enlarged, the character of the projecting portion, whether smooth or uneven. In infants and children the region of the gall-bladder is palpated, but it is difficult to determine in these subjects whether this organ is enlarged or absent. Henoch and Murchison have recorded fatal cases of increasing and persistent icterus in which there was congenital absence of the gall-bladder.

Percussion.—Percussion should be performed in the mid-line from the base of the xiphoid cartilage downward, in the right mammillary line from above downward, and sometimes in the mid-axillary line. In order to determine accurately the superficial dulness, the whole extent of the dulness should be measured. This is rarely necessary except in investigations for scientific purposes. In cases of effusion into the pleural cavity, the upper limit of dulness is continuous with the dulness or flatness of the fluid. The displacement below the border of the ribs only can then be determined. In rare cases of subphrenic abscess there is an extension of the upper limit of dulness into the limits of the chest cavity, and displacement of the lower border of the liver downward. Steffen gives the following measurements of the superficial liver dulness in the median and mammillary lines:

FIG. 101.



Method of palpating the projection of the liver below the ribs.

	Midline.	Mammillary line.
At birth	3.5 cm.	2 cm.
At one month	5 "	5 "
At six months	4.5 "	4.5 "
At one year	4.5 "	4 "
At two years	5.2 "	5 "
At five years	5 "	6.5 "
At ten years	5 "	6 "

These measurements also vary greatly, especially in infants under one year of age.

The following tumors and conditions simulate enlargement or disease of the liver: phantom tumor; circumscribed empyema, or pleuritic effusion; subphrenic abscess; circumscribed peritoneal effusion between the liver and diaphragm ; tumors or cysts of the right kidney.

PHANTOM TUMOR is described by Murchison. It is a soft or hard epigastric tumor, which may project downward as far as the umbilicus. Whether it is dull with a tympanitic note, or tympanitic, depends on the amount of muscular contraction. There is no fluctuation or flatness. The tumor is present when the patient is standing or in the recumbent position. It disappears under anæsthesia. A tumor of this kind should not be punctured until it has been observed under anæsthesia, since there is danger of puncturing the intestine and causing peritonitis.

EMPYEMA.—In simple or encapsulated empyema on the right side, the liver is displaced downward. The upper dulness extends into the pleural cavity ; the lower part of the thorax may enlarge to such an extent as to press the ribs apart and cause fluctuation between them. There will be dulness or flatness in front or behind over the lower part of the pleural space, and perhaps disappearance of the respiratory murmur. It should not be forgotten that there is always a possibility of the presence of subphrenic abscess, or of abscess in the upper part or on the surface of the liver, bulging into the pleural cavity. In that case there will not only be bulging of the lower ribs, but also a continuation of dulness for a variable distance upward. The liver may be enlarged downward or not at all. If the tumor is beneath the diaphragm and displaces the liver downward, the respiratory murmur may be heard to the normal, or almost normal, limit, and yet dulness due to the upward projection of the tumor may be present.

KIDNEY TUMOR may extend from behind, beneath the liver, and simulate liver tumor. In such cases, the lumbar flatness extending below the border of the ribs will be a guide.

ENLARGEMENTS OF THE LIVER.—Enlargements of the liver in infancy and childhood present much the same physical signs as in the adult, but there are some states which are peculiar to early life.

ANÆMIA INFANTUM PSEUDOLEUKÆMICA OF VON JAKSCH causes great enlargement of the liver and spleen. The lower edge of the liver is rounded ; the lymph-nodes are enlarged, and the blood presents certain features characteristic of this anæmia.

SIMPLE RACHITIS causes slight or marked enlargement of the liver, as well as real enlargement of the spleen. In some cases, the liver is not really enlarged, but may be displaced downward by the deformity of the thorax. Simple icterus usually causes enlargement of the liver, which retrogrades after a few weeks.

CONGENITAL SYPHILIS may cause slight enlargement of the liver which, up to the end of the second year, is present without icterus. The liver is enlarged in cirrhosis, abscess, and fatty degeneration of the organ. It is greatly enlarged in acute and chronic leukæmia.

Jaundice.

(Catarrhal Icterus; Catarrhal Jaundice; Infectious Icterus.)

Simple jaundice is a common disease of infancy and childhood. In its simplest form, it was formerly believed to be due to an obstruction of the common bile-duct with mucus. In recent years, the French clinicians have described a form of jaundice which they regarded as infectious. The first cases of the kind were published in 1881 by Weiss, Chauffard, and Landouzy, in France, and by Weil, in Germany. There is at present a tendency to regard all cases of jaundice in infants and children, not due to mechanical obstruction of the duct or disease of the liver, as infectious (Botkin, Hennig, Barthez, Henoch, and others). Thus simple icterus would be regarded as a mild form of infectious icterus. This view has recently been elaborated by Kissel. The theory, that errors of diet cause a catarrh of the gut, extending into the duct and thus obstructing it, finds little support. On the other hand, the theory of the infectious nature of even the mildest cases of jaundice is supported by the fact that these cases occur in groups and epidemics.

Morbid Anatomy.—In cases of fatal icterus, there are found atrophy and fatty degeneration of the liver cells. The interstitial tissue around the portal vein is infiltrated with small round cells. There is parenchymatous degeneration of the kidney. The whole picture resembles that of acute yellow atrophy. The mild cases of icterus have not yet been studied.

Bacteriology.—The bacteriology of the severer form remains to be studied. In one case Jäger found a bacillus of the proteus group in the urine.

Occurrence.—The disease may appear at any period of infancy and childhood. It is most common between the second and fifth years.

At present, all primary forms of jaundice may be clinically classified as follows: The very mild forms (catarrhal icterus); the severer forms; the fatal forms. It is highly probable that all are infectious in origin. The secondary forms of jaundice are not considered in this section.

Symptoms.—In the mildest forms there are no symptoms at the onset. In some mild cases there are vomiting and fetor of the breath, and the tongue is coated. The skin assumes a saffron hue and the conjunctivæ are distinctly yellow. The appetite is capricious; the urine is brownish and contains bile-pigment. The movements are like clay, and may have a bad odor. There is pruritus of the surface. The child may be somewhat depressed. In the very mild forms there is no febrile movement. In the majority of cases, there is rapidity of pulse and, in some cases, irregularity. In the severer forms the symptoms are more marked. The vomiting recurs at intervals, the intensity of the jaundice is much the same as in the mild forms, and the temperature may in the course of the disease be raised a degree or more. The attack may be ushered in by a chill. There is some prostration and, in a few cases, diarrhœa. The fatal cases, which were first described by Weiss and the French school, are severer forms of infection. The symptoms of cholæmia are much more marked. There are delirium, unconsciousness, and cerebral symptoms. The pulse is greatly increased and the respirations are irregular. The patients die in an asthenic state.

The liver is enlarged in even the mildest forms. In a recent series of 20 cases of mild icterus, I found the liver enlarged from four to seven centimetres below the border of the ribs, in the mammillary line. The spleen was enlarged in most cases. The fact that in the mildest forms there is enlargement of the spleen lends support to the infectious theory of the disease. In the majority of my cases, the liver remained enlarged long after the icterus had disappeared. Kissel also found this to be the case. In some cases, three months elapsed before the liver returned to the normal limits.

Duration.—The disease, even in the mild form, lasts from two to three weeks. The fatal forms may run their course much more rapidly.

The **treatment** of icterus is very simple. An initial dose of calomel is given and the bowels are well evacuated. The patient is put on a milk diet, and is given a daily enema of water at a temperature of 85° F. (29.4° C.). On every second day a small dose of calomel, grain ½ (0.03), is given to aid the enemata. Fresh air and daily alkaline baths are beneficial. Alkaline baths are made by adding a few tablespoonfuls of sodium carbonate and an equal quantity of salt to the water.

Congenital Obstruction of the Bile-ducts.

The **etiology** of this affection is obscure. Some 70 cases of this condition were recently collected by Morse from the literature. The infants may be apparently normal at birth. Intense jaundice is the first symptom noticed at birth, or on the second to the fourth day after birth. Meconium is first passed by the infant, and then the stools are clay colored. The urine contains biliary coloring-matter. The liver is enlarged, as is also the spleen. Hemorrhages from the stomach and intestine and into the skin occur in time. Death occurs early, or in from three to eight months.

Morbid Anatomy.—Some portion of the bile-ducts may be obliterated and replaced by connective tissue. In other cases the walls of the ducts are simply swollen. The liver is enlarged and the seat of cirrhotic changes.

Cirrhosis of the Liver.

This disease is rare in infancy and childhood. Of 62 cases collected from the literature by v. Kahlden, 5 occurred in the newborn, 12 in the first two years of life, and 28 from the ninth to the thirteenth year. It is more prevalent in the male sex. Of those cases in which the size of the liver was recorded 19 were atrophic, 15 hypertrophic, and 6 normal in size.

Etiology.—Demme has published 2 cases in children addicted to the use of alcohol. The influence of heart disease and the infectious diseases, such as scarlet fever and measles, in causing cirrhosis of the liver is not as yet understood. Cirrhosis of the liver occurs in forms of peritoneal tuberculosis and in syphilis.

The **morbid anatomy** of the affection is the same as in the adult.

The **symptoms**, which are the same as in the adult, include enlargement of the liver and spleen, icterus, and ascites. The icterus is, as in the adult, constant.

The liver is not always enlarged, and in the cases in which it is of normal size the difficulties of diagnosis are increased. The spleen is most constantly enlarged.

The recorded cases of cirrhosis following or complicating the exanthemata and diphtheria gave no previous symptoms.

Fatty Degeneration of the Liver.

Fatty degeneration of the liver, with or without enlargement of the organ, occurs in forms of subacute and chronic constitutional dyscrasia. I have seen this disease in infants who died with tuberculosis, chronic or subacute intestinal diarrhœa, rachitis, Henoch's purpura, or acute leukæmia. I have also seen it in cases of phosphorus-poisoning. The symptoms and signs do not differ from those seen in the adult. The diagnosis can hardly be made during life.

Syphilis of the Liver.

Enlargement of the liver is common in syphilis of infants and children. The spleen may also be enlarged. There may be icterus. There may be other symptoms of syphilis, but none which can be traced to enlargement of the liver.

There are four histological forms of this variety of hepatic enlargement:

a. The form in which gummata are found in the liver. This is rare. I saw a case in an infant sixteen months of age in which there were also gummata of the cranial and the long bones.

b. The diffusely cirrhotic liver. In this form the connective tissue is quite evenly distributed throughout the liver.

c. The lobulated liver, in which the connective tissue divides the organ into sections. I have seen a case in a girl eight years of age.

d. The so-called miliary syphilis of the liver, in which the organ is strewn with miliary collections of round cells closely resembling miliary tubercle. The nodules are situated in the interstitial connective tissue. They rapidly undergo fatty degeneration.

Clinically the cases which I have met were mostly those in which the liver, hard and nodular, could be felt below the border of the ribs. In one case there was a history of syphilitic accidents, in another old cicatrices existed on the lips and face. In a third case the patient had Hutchinson teeth; the liver and spleen were both enlarged and nodular.

Abscess of the Liver.

(*Suppurative Hepatitis.*)

This disease occurs in the newborn as a form of sepsis. Otherwise its etiology in infancy and childhood is identical with that in the adult. It may follow a traumatism or complicate appendicitis (septic), it may occur in peritonitis with pyelophlebitis, or it may follow the infectious diseases. In the literature rare cases are described, in which Ascarides lumbricoides have caused abscess of the liver in children, by migrating into the gall-bladder through the common duct.

The occurrence of this disease, though not rare in tropical countries, is less frequent in districts in which dysentery is not endemic. It may occur as early as the fifth month of infancy (Oliveira). The left lobe of the liver is most frequently involved. The Amœba coli is not always the cause, being an etiological factor in 20 per cent. of the cases. The **symptoms** in these cases are first those of dysentery; then, after improvement sets in, the symptoms of abscess, with fever, swelling of the abdomen, and enlargement of the liver upon palpation appear. The liver may enlarge as much as 10 cm. below the tip of the ensiform cartilage.

Course.—The abscess may perforate into the gut, pleura, or peritoneum. If it perforate into the gut recovery results. Any other termination is disastrous.

The **treatment** of abscess of the liver in children is much the same as in the adult. If operated early the prognosis is good.

Acute Yellow Atrophy of the Liver.

The disease is extremely rare in infancy and childhood. **Lanz** published a case in a boy four years of age. In that there was no splenic tumor or hemorrhages, it differed from the picture in adult cases. The cases in the literature are as follows: Pollitzer, infant, one month of age; Senator, infant, eight months; Mann, infant, ten months; Greves, infant, twenty months; Widerhofer, child, one and three-fourths years; Rehn, child, two and one-half years; Löschner, child, three and one-half years; Mettenhemier, child, four years; West, child, six years; Merkel, child, six and one-half years; Rosenheim, child, ten years; Steiner, child, ten years; Folwarczny, child, fourteen years.

I have seen only one case of atrophic liver. The patient, a boy of eleven years of age, with very small kidneys, had nephritis which had appeared six years after an attack of scarlet fever. The liver dulness became gradually smaller from the time of admission to the hospital until death. At autopsy, the liver was found to have one-half the normal weight and to be the seat of marked parenchymatous degeneration.

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VII. DISEASES OF THE PERITONEUM.

Ascites.

Ascites is a serous effusion into the peritoneal sac, and, as in the adult subject, it is generally secondary either to some disease of the peritoneum, such as tuberculosis, or chronic disease of the heart, liver, or kidneys. It may also be due to some obstruction of the portal circulation, caused by enlarged glands or tumors of the peritoneum. Ordinary ascites has the same characteristics in the infant and child as in the adult, and is recognized by the same physical signs. It is therefore superfluous to go into details in this place as to the physical characteristics of the fluid accumulation in the peritoneal cavity of infants or children.

Some rare forms of ascites may be congenital. In diagnosing ascites in infants and children, we must be careful not to confound it with local accumulations of fluid due to cysts or tumors in the peritoneal cavity. Cysts, or cystic tumors, have local circumscribed physical characteristics, and with care they cannot be mistaken for ascites. There is a form of ascites which occurs rarely in children, and of which I have seen one example in a boy six years of age. It is called chylous ascites, and is marked by its chronicity and the milky or fatty nature of the exudate. It is more frequent in adults; but when present in infants or children, it is found between the ages of seven and ten years. In one case recorded by Wicklen, the accumulation followed an attack of pertussis in an infant six months of age.

The **etiology** of chylous ascites is obscure, although in some cases tuberculosis of the peritoneum has been found post mortem. It has followed traumatism, eruptive fevers, or an infection with filaria. The symptoms are those of ascites, and it is not until the withdrawal of the fluid that the true nature of the affection is discovered. The fluid withdrawn has a milky, opalescent appearance, and is of two forms, in one of which there is a fine emulsion of fat-globules with red and white blood-cells; the other form contains no such element, but is chylous in color. At autopsy various lesions have been found, as stated, including tuberculosis, syphilis of the liver, cirrhosis of the liver, an enlarged spleen, with lesions of the thoracic duct. In some cases there has been tuberculosis of the thoracic duct, or this combined with tuberculous disease of the lymph-nodes, with apparent obstruction of the lymph-vessels.

The **treatment** of ascites in children is carried out along the same lines as in the adult patient.

Acute Peritonitis.

Acute peritonitis may be general or local, and is due to an infection of the peritoneum.

Etiology.—According to Tavel, Lanz, and Treves, the disease is caused by various bacteria, such as streptococci, staphylococci, pneumococci, or coli bacteria, but the most active rôle, even in the traumatic and perforative forms, is played by the Bacterium coli communis. Krogius examined 40 cases of perforative peritonitis following appendicitis, in 20 of which he found two or three species of bacteria; in only 7 cases did he find Bacterium coli alone. The species found were generally coli bacteria in combination with diplococci, pneumococci, Diplococcus intestinalis, streptococci, coli gracilis. The remaining cases contained the Streptococcus pyogenes, pyocyaneus, and Proteus vulgaris. The coli, however, was the most frequent microörganism found. It is to be remarked that in 21 cases the Diplococcus pneumoniæ was found combined with the Bacterium coli. This form must not be confounded with the cases in which the pneumococcus is found as the causative agent of peritonitis, especially in children (Netter, Sevestre, and others).

We may have: (1) Acute tuberculous peritonitis. (2) Perforative peritonitis, due to traumatism or some pathological perforation of the viscera or the serous coat of the intestine as a result of tuberculosis, typhoid fever, dysentery, perforating ulcer of the stomach or duodenum, abscess of the liver, cyst of the liver, kidney, or spleen, rupture of the gall-bladder, strangulated hernia, intestinal intussusception, appendicitis, perforating lumbricoides—all these can be accompanied by the escape of gas, fæcal matter, bile, or blood into the peritoneal cavity. (3) Peritonitis may take place by extension, as is observed in cases where inflammation extends from a viscus without perforations. (4) Peritonitis may occur as the result of traumatism, as a blow or fall or an operation. (5) Pneumococci may cause an acute primary peritonitis, or may give rise to the affection by extension from the pleura or lung. (6) There is a gonorrhœal form of peritonitis. (7) Peritonitis may occur in the fœtus or in the newborn. The latter has been described by Billard as following intrauterine infection, as a result of maternal disease; or in the newborn peritonitis may be caused by streptococcal infection of the umbilicus, and extension from this point to the peritoneum.

The **symptoms** of acute peritonitis at the onset may be insidious. Such forms occur in cachectic, marantic infants, or children; or the onset may be acute and sudden, as in the primary form.

Pain may be localized either in the iliac fossa or around the umbilicus, spreading thence over the whole abdomen. The child lies quietly on the back, with superficial respiratory movement. There is, as in the adult subject, meteorism or tympanites. There is vomiting, first of the contents of the stomach, then the vomitus becomes green or biliary. It may subside after two or three days. There may be a diarrhœa, but in most cases there is constipation as obstinate as in intestinal obstruction. The tongue is moist, then dry; the buccal mucous membrane may be covered with sprue; the urine may be suppressed, and, as in the adult, there may be facies. The pulse ranges from 120 to 150, small and thready. The fever varies in extent, depending very much on the acuity of the infection. In perforative peritonitis there will be a sharp rise of temperature.

The physical signs are much the same as are found in the adult. There is tympanites, the abdomen is distended, there is a disappearance of the liver dulness. In localized peritonitis there is local pain; in general peritonitis the pain is general. If the peritonitis becomes general, there is, as a rule, an accumulation of fluid in the peritoneal cavity, and this may be made out by dulness in the flanks. As a rule, an examination of the blood will reveal an increased number of leucocytes or so-called leucocytosis, especially in the perforative forms. This latter sign is not of much value unless a previous leucocyte-count has been made or the case has been under constant observation, such as in forms of perforation occurring in typhoid fever, for even in these cases the increase in the number of the leucocytes is only comparative. Thus the leucocyte-count in the course of typhoid fever may be 6000 to 8000; whereas after perforation the leucocytes may not increase beyond 10,000 to 12,000. In other words, they may simply reach the normal limit.

Course and Termination.—Acute peritonitis, as in the adult, may remain localized or may spread and become general. In the latter case the prognosis is very grave. If local the exudate may become encysted, or, if general and left to itself, may result fatally, or the exudate in the peritoneal cavity may rupture in the vicinity of the umbilicus or through the vagina or rectum. Foudroyant cases last two or three days and result in death. This is especially so of the newborn.

Among the **complications** of acute peritonitis, either general or localized, are pleurisy, pericarditis, meningitis, pyæmia.

Prognosis.—As stated, the general perforative forms present the gravest prognosis. Peritonitis of the newborn is fatal.

Differential Prognosis.—Peritonitis, acute, localized, or diffuse, must be differentiated from typhoid fever. In the latter disease there is sometimes a severe inflammation in the vicinity of the vermiform appendix, and in such cases we should be very careful that a perforation has not escaped our notice.

Colprostasis, or intestinal invagination, and gastro-enteritis may be mistaken for appendicitis, especially in young children, if the meteorism is great.

Gonococcal Peritonitis.

Gonococcal peritonitis results from an infection of the peritoneal cavity by the Gonococcus of Neisser. Comby records 7 cases of gonococcal peritonitis. Hunner and Harris record 7 cases. I have seen 2 cases. The infection takes place by way of the uterus and Fallopian tubes in the majority of cases.

Etiology.—As has been stated, the gonococcus is the etiological factor in these cases, and the majority of recorded cases in children have occurred in young infants and children suffering from vulvovaginitis of a gonorrhœal nature. In my two cases this was the etiological factor. The symptoms are sudden pain, vomiting, fever; or in other cases there results in the course of the vaginitis severe pelvic pain. In some cases the pain and fever are of short duration, and it must be surmised in these cases that the inflammation remains well localized to the pelvis. I have seen quite a number of these cases complicating vaginitis in young girls. The French have given the name of *peritonism* to these cases, thereby wishing to indicate their benign nature. The symptoms are so slight that one can scarcely believe that inflammatory reaction is present. Baginsky has published a case of general peritonitis resulting from gonorrhœa of the tubes, with an abscess-formation in Douglas's pouch. The gonococcus of this form of peritonitis may be associated with other bacteria, such as the staphylococcus. There are several forms of gonorrhœal peritonitis: the general acute form, ending in death; the benign pelvic form, with subumbilical pain; and a third form occurring as a pelvioperitonitis with adhesions and salpingitis. Diagnosis must be made from appendicitis, for which it may be mistaken. Given a case of gonorrhœal infection of the genitals in children, with sudden abdominal pain, fever, and general abdominal distention, the diagnosis presents no difficulties.

Prognosis.—The French writers insist that the prognosis of gonorrhœal peritonitis is benign. On the other hand, such a prognosis will depend very much on the severity of the infection. Inasmuch as I have personally seen three fatal cases found at autopsy to have been due to gonorrhœal peritonitis complicating vulvovaginitis, I cannot regard the general form of gonorrhœal peritonitis as anything but a grave infection particularly fatal to children. The treatment of gonorrhœal peritonitis varies according to the extent of the infection. If the peritonitis is localized to the pelvis it is quite evident that the treatment should be mostly on the lines laid down for the adult subject. If the peritonitis becomes general there will be a difference of opinion as to whether surgical interference is necessary. It is not within the scope of this work to discuss this aspect of the subject; but in an excellent résumé of the subject by Hunner and Harris the surgical interference in gonorrhœal peritonitis is rather discouraged. In general peritonitis of gonorrhœal nature rest in bed, hot turpentine-stupes alternating every hour with warm stupes, mild catharsis, liquid diet, hydrotherapy, and general medical treatment are rather to be advocated.

Pneumococcal Peritonitis.

Pneumococcal peritonitis, as has been stated, may be primary, and as such occurs most frequently from the second to the twelfth year of life. It may be secondary to pulmonary disease, such as pneumonia or pleurisy; or may be primary, resulting from an infection of the peritoneum either through the blood or the genitals. The frequency of encapsulation of the pus around the umbilicus makes the genital way of infection very probable.

The **course** of the symptoms in this disease recalls that of a pneumonia, by its sudden onset in subjects previously in good health. There is a chill, followed by fever, pain, vomiting, and some diarrhœa. After a period of eight days there is a defervescence of the fever and abatement of the symptoms. The abdomen, which has been previously distended and generally painful, with all the physical signs found in other forms of peritonitis, remains large and distended, pus accumulates, the umbilicus becomes prominent, and in this way we have a picture resembling ascites or tuberculosis of the peritoneum. I have seen a case in which the latter diagnosis was made. Pus may break spontaneously at the umbilicus or perforate through the vagina. The disease is more frequent in girls than in boys, and, as has been stated, the pus has a tendency to become encysted and discharge at the umbilicus. The pus is of a creamy, yellow color, without odor.

Michant has collected 33 cases of pneumococcal peritonitis occurring in children: 27 of these were girls; 22 were encysted, 11 were generalized. In 27 cases the disease was primary.

The prognosis, as a rule, is good, for in most of the cases, the pus being encysted, the general peritoneal cavity remains free of infection. In the general form, however, the prognosis is more grave. Of 11 cases of this form 9 died of sepsis. This form of peritonitis is naturally mistaken for peritonitis following appendicitis. It may be distinguished from the latter, however, by its benign course. The pus, if it becomes encysted, may distend the abdomen to an enormous extent. I have seen a case in which the distention of the abdomen was enormous, resulting in the obstruction of the portal circulation, with dilatation of the superficial abdominal veins. There was perforation at the umbilicus, and a discharge of several pints of pus, followed by recovery of the patient. Appendicitis is more acute in its nature and does not extend over such a long period of time, with the benign result, as seen in this form of peritonitis.

Tuberculosis of the peritoneum can hardly be mistaken for this form of peritonitis. Given a distention of the abdomen by a fluid pointing at the umbilicus, which fluid is found to be pus, we may surmise that there is a pneumococcal peritonitis. A positive diagnosis can only be made by bacterial examination of the pus.

Simple Chronic Peritonitis.

Although Henoch and Müller have reported cases of chronic idiopathic non-tuberculous peritonitis, its occurrence is still a matter of dispute. Nothnagel, Unger, and Heubner, while not denying *in toto* its possible occurrence, insist on its extreme rarity. The absence in these cases of progressive emaciation is no proof of the non-tuberculous nature of the affection. The absence of the tubercle bacillus in the abdominal exudate is of slight diagnostic value. In 29 cases of undoubted tuberculosis of the peritoneum Herzfeld found the bacillus only once in the ascitic fluid. In some forms of tuberculous peritonitis the nutrition may not only be good, but there may be no history of heredity or scrofulosis. It is manifest that under these conditions it is impossible to describe a disease the existence of which is still in doubt.

SECTION VI.

DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE AND NASOPHARYNX.

EXAMINATION of the nose in infants and children consists first in a general inspection of the organ. In this way any congenital deformity, particularly of the septum nasi, is noted. Some forms of congenital syphilis carry with them a malformation of the bony septum, by which the bridge of the nose is markedly depressed in very much the same manner as that of the adult. Deviations of the bony septum are sometimes indicated by an angular deflection of the organ to one or the other side. The interior of the nares may be inspected, as in the adult, by elevating the tip of the nose upward and backward, or by means of small-sized specula. One of the most useful methods with the author of discovering any obstruction in the nares, especially in the newborn and young infant, in whom instruments, such as specula, cannot be applied, is the passage of a small probe into the nares in a backward direction. This procedure is painless, and in the majority of cases will suffice to discover any swelling of the mucous membrane or bony obstruction, if such be present. The introduction of the index finger of one or the other hand into the nasopharyngeal space for the purpose of palpating the walls of this structure has been dilated upon elsewhere in discussing adenoids. It must be reiterated here that this procedure should be carried out with the utmost circumspection and gentleness. The finger should be scrupulously clean, and the finger-nail carefully trimmed, so as not to cause undue traumatism. In older children the inspection of the posterior nasal space by mirror, if this is possible, is much to be preferred to the digital examination.

Acute Nasal Catarrh.

This is a common affection of infancy and early childhood. In the newborn it follows as a direct result of exposure combined with infection, either by the lochia of the mother or uncleanliness of the bath water. In older infants and children acute coryza occurs sporadically or in epidemic form. It is apt to be seen at certain seasons of the year—early spring or autumn—when children are subjected to sudden changes of temperature of the outer air and that of the living apartments. Infection by bacteria plays a leading rôle in this disease, as in other affections of the nasopharynx. Infants and children are apt to be infected by adults around them, especially careless nurses. One child may infect the other, or acute nasal catarrh may usher in the acute infectious diseases, such as measles, bronchitis, influenza, pneumonia, pertussis, and in some cases cerebrospinal meningitis. One attack of nasal catarrh may lead to another, and thus, in the end, to chronic nasopharyngeal catarrh. Some infants and children have a tendency to contract coryza upon the least exposure to a cold or dust-laden atmosphere. Such infants and children are pale or rachitic or show some constitutional dyscrasia, such as lymphatism.

The **symptoms** of acute nasal catarrh, or coryza, consist in a slight discharge of a serous or seropurulent secretion from the nostrils. This discharge may be thin or mucoid in consistence, and may be small in quantity, occur in the early morning, but slight in amount during the day. There is, as a rule, but little or no febrile disturbance in mild cases. In the severer types there may be involvement of the lachrymal ducts, with slight or marked redness of the conjunctiva, orbital and palpebral. In the latter form there is lachrymation and photophobia, with or without slight febrile movement. In other cases the infants or young children are uneasy, do not take their usual day naps or their food, and have a slight cough. An inspection of the fauces may reveal but little inflammatory reaction, and the tonsils may be enlarged to a slight degree. As a rule, all these organs are drawn into the picture. In some cases conjunctivitis may be the first symptom, and the nasopharyngeal catarrh may follow. Restlessness in some cases and temporarily high temperature are explained by an inspection of the drumhead of the ears, which may be slightly or even markedly red without bulging of that structure. In other words, a myringitis may give rise to a temperature of short duration simulating a true otitis.

Course.—As a rule, the affection is self-limited, lasts two or three days, and then retrogrades; in other cases the physician is annoyed at the persistence with which certain symptoms continue and refuse to recede with therapeutical measures. When the symptoms are apparently subsiding the temperature may suddenly rise to 104° F., and this in the face of the most trivial physical signs. In such cases an inspection of the ear-drum may reveal a slight or marked otitis. In the nursing infant the obstruction and swelling in the nose may cause difficulties in nursing, and the bowels may show slight evidences of infection, caused by the infant swallowing the discharges from the nasopharynx.

Diagnosis.—This is not difficult, but in the face of any coryza of an acute type the patients should be examined as to the presence or absence of measles, bronchitis, pneumonia, or otitis, especially if a temperature of a high intermittent type is present after the second day of the disease.

The **prognosis** is good, but it is not invariably so, as to a rapid termination in an uncomplicated recovery. During the early spring an acute coryza is not infrequently followed by an otitis, which may be catarrhal, purulent, or even eventually involve the mastoid. We should therefore not regard lightly any coryza in an infant if the symptoms persist beyond the third day, and other organs, such as the ears or bronchi, become involved.

Treatment.—The mild forms of acute nasal catarrh in infancy and childhood are self-limited as to duration, and simple cleanliness with nursing will in most cases suffice in the treatment of the disease. In infants the nose should be carefully cleansed with a spud of cotton after the morning bath, and then a drop of castor oil allowed to flow back into the nares. If this one application does not suffice to keep the nares clear of secretion, and nursing is difficult on account of the accumulation of secretion in the nares, this procedure must be repeated during the day. Mild cases need no medicinal treatment. If the throat is drawn into the picture, a small dose of 2 or 3 minims of the tincture of the chloride of iron combined with glycerin may be given every three hours. If there is much conjunctivitis a saturated solution of boric acid flushed in the eye two or three times daily is, as a rule, efficient; and in the subacute stages of the conjunctivitis a drop of a solution of sulphate of zinc, 2 grains to the ounce, may be instilled into the eye once or twice daily. The application of a copper pencil to the conjunctivæ once in the subacute state is advised by some, though the author thinks that such procedure should be carried out by the oculist. The reaction which follows the application of copper subsides soon, to leave the conjunctivæ in a less angry condition. If an inspection of the ear-drum shows a redness without bulging of the drumhead, we may temporize, and if there is reason to believe that pain is present, a drop of warm hyoscyamus oil instilled into the ear once or twice daily will give relief. Marked otitis requires more elaborate treatment, which should be carried out by the otitic specialist.

Sprays and douches are not applicable to infants and young children, on account of the resistance made by these patients to any attempts of this kind. Only older children can be taught to gargle or spray. Where this is possible a mild solution of listerine or Dobell's solution is all that is called for. I have never found it necessary to use the so-called coryza tablets, which contained stronger drugs, in children. If temperature is not present, the open air is certainly not contraindicated; on the contrary, it may cut short a rebellious catarrh.

Chronic Nasal Catarrh.

This is a condition found in infants and children, as a result of repeated attacks of acute nasal catarrh, in a constitution undermined by a pre-existent dyscrasia, such as lymphatism. In most infants and children this tendency to chronic catarrh is hereditary. There are evidences in these little ones of similar conditions elsewhere. Such infants and children may suffer from forms of conjunctivitis. Keratitis, dermal eczema, or eruptions of various kinds, anæmia, adenoids, nasal polypi, deviated nasal septum—may be combined with hypertrophy of the nasal mucous membrane. A chronic nasal discharge is present, and with it erosion of the nostrils and a distinct odor to the breath (ozæna). The tonsils in this stage are enlarged. Foreign bodies may set up a chronic inflammatory condition of the nares in children suffering from chronic catarrh; this fact must never be lost sight of.

Symptoms of chronic nasal catarrh are combined with certain chronic hypertrophic conditions of the throat and nasopharynx. Thus, very young infants, unless they are subjects of syphilis or adenoids, are not chronic sufferers from nasal catarrh. There is then a constant discharge from the nose and the nasopharynx. In older children, from five to eight years of age, the hypertrophy of the nasal mucous membrane and the nasopharynx results in a profuse mucopurulent secretion in the pharynx and nose. These children are constantly coughing, and are subject to repeated attacks of so-called cold, the tonsils being enlarged, the lymph-nodes at the angle of the jaw are also enlarged, as also the nodes of the neck behind the sternomastoid muscle, and elsewhere in the body. The alæ nasi are thickened and reveal erosions. The lips are also thickened as the result of obstructed circulation. Breathing is mostly oral. An inspection of the fauces shows the posterior pharyngeal walls coated with mucopus and studded with hypertrophied structures made up of lymphoid tissue called follicles. In older children these symptoms may be combined with symptoms of atrophic rhinitis, in which the mucous membrane of the nose loses its hypertrophic appearance and becomes thin, atrophic, and coated with dry greenish crusts. Instead of obstruction there is found a wide nasal passage, and there is distinct odor to the breath and nasal discharges. There are forms of chronic nasal catarrh in which the above symptoms are present to a very mild degree. Thus, with the nasal catarrh there are enlarged tonsils and a few adenoids, and only an occasional odor to the breath. This condition is found in children who have been treated with indifferent success. The very marked cases of nasal catarrh in lymphatic subjects may be combined with a conjunctivitis of a chronic type or granular lids and eruptions, such as ecthyma and pustular eczema of the chronic type, all of which indicate the presence of a dyscrasia.

The **treatment** of the above conditions are first local; the tonsils and growths in the nasopharynx must either be removed or treated locally. The minutiæ of such treatment belongs to the realm of nasal specialism. The local treatment must, however, be combined with general constitutional hygiene and treatment. The remedies best suited to the conditions above are discussed under the heading of Scrofulosis and Lymphatism.

Diphtheritic Rhinitis.

An apparent simple rhinitis of a catarrhal character may in a short time take on the characteristics of a diphtheritic process, due to an infection with the Klebs-Löffler bacillus. There is a profuse seropurulent or serosanguinolent discharge from the nose, with shreds of pseudomembrane, erosions of the nares, and extension of the membrane backward to the nasopharynx and downward to the larynx. This true diphtheria is accompanied by the glandular swellings and constitutional symptoms characteristic of the disease elsewhere. On the other hand, there is a form of rhinitis called pseudomembranous rhinitis, in which the disease remains fairly limited to the nose. There are two forms of pseudomembranous rhinitis, the truly diphtheritic form, in which the Klebs-Löffler bacillus is an etiological factor, and the streptococcal form, both of which have a similar symptomatology. The form of disease to which we refer is mild in its course, and begins like a catarrhal rhinitis, but on the third day a white coating is formed over most of the inflamed area; that is, on the turbinated bodies and the septum of the nose. This coating, which is pseudomembrane, cannot be either washed off or wiped away with absorbent cotton, but may be peeled off with the forceps. As soon as the membrane is removed, however, it reforms; it is dead white and opaque, and firmly attached to the parts beneath, and, when detached, considerable violence must be used, and a bleeding surface is left.

In some cases casts of pseudomembrane may be removed from the nostrils. Chapin, Bresgan, Schuler, Hartmann, and Muldenhauer have all described these cases. This membranous condition lasts in some cases from twelve to fourteen days, and though, as has been intimated, some of them must be looked upon as true diphtheria, the prognosis is generally good. In the streptococcal cases the prognosis also is good. We must never forget, however, that though there is in a certain proportion of cases of membranous rhinitis very few constitutional symptoms, and very little tendency of the disease to spread downward from the nasopharynx to the larynx, these cases should always be examined for the presence of the Klebs-Löffler bacillus, and if found should be treated as a diphtheritic process.

Foreign Bodies in the Nose.

Children are prone to put beans, buttons, pins, and foreign bodies of all kinds into their noses. These foreign bodies at first cause little disturbance; after awhile, however, they become a source of pain and irritation, and, if not discovered, chronic nasal catarrh, ulceration, and even abscess may result. The removal of foreign bodies from the nose in many cases requires nothing more than ordinary skill. Some children can be taught to blow the foreign body out of the nostril by occluding the unobstructed nostril with the finger. In other cases the foreign body can be removed with the forceps. In the third set of cases, a scoop introduced into the nostril so as to hook the body posteriorly is an efficient means to remove it; in other words, a bent probe or buttonhook.

Epistaxis.

Epistaxis is rare in the newborn, except as a manifestation of syphilis or sepsis. In infants and children it may be caused by traumatism of any kind, and is seen mostly in school children who have been confined in warm rooms and have developed nasal catarrh with or without adenoid vegetations. There may be in these cases small ulcers or erosions of the septum nasi. Epistaxis occurs in the course of acute or chronic rhinitis, typhoid fever, pneumonia, infectious diseases, diseases of the heart, chlorosis, hæmophilia, scurvy, morbus maculosus, and finally, it occurs in young girls entering on the period of menstrual activity. It may occur in these subjects also as a vicarious form of menstruation. Epistaxis, as a rule, is unaccompanied by any symptoms other than those of the bleeding, in drops, from the nose. In very few cases does this hemorrhage become alarming unless there is a history of hæmophilia. The quantity of blood lost is often exaggerated by the patients, and rarely exceeds an ounce. Nasal hemorrhage may occur daily, or it may recur every few days or weeks, in which case there is always a suspicion either of traumatism, such as picking the nose, or a chronic nasal catarrh. Some children complain of dizziness or vertigo preceding the attack. Others become greatly alarmed by the sight of blood. Children below the age of three or four years rarely have epistaxis except as a result of traumatism or nasal ulceration. In some cases hemorrhage is really alarming, amounting to a rhinorrhagia. In these cases there is a suspicion of dyscrasia; in many cases blood may during sleep flow down the posterior nares into the œsophagus and stomach, and after a time the clotted blood may be vomited or passed in the movements, thus simulating hemorrhage from the stomach or bowels, and in young infants melæna.

Adenoid Vegetations.

It is not within the scope of this work to give more than the diagnostic bearings of these growths as they occur in infants and children. On inspection, the posterior nasopharynx in the normal infant is frequently seen to be the seat of more or less adenoid tissue. The diseased condition is simply an exaggerated growth of the tissue which is normally present in this space. Clinically there are three distinct classes of cases that suffer from adenoids:

The first class comprises those in which the adenoids cause few or no symptoms. The children when in good health breathe through the nose and keep the mouth closed during sleep. They are peculiarly susceptible to slight colds or catarrh, and when thus affected the tonsils enlarge, the nose becomes obstructed by secretion, there is difficulty in breathing, and the patient sleeps with the mouth open. On the subsidence of the inflammatory condition the normal status is re-established. The children are subject to recurrent attacks of tonsillitis, and with each recurrence the symptoms of adenoids become more marked. The patients contract obstinate coughs which resist all treatment, and epistaxis occurs from causes apparently trivial.

The second class of cases comprises those in which, in addition to the enlarged tonsils, there are enlarged lymph-nodes in various regions of the body. The patients are pale and present all the symptoms of lymphatism. Their voices have a nasal intonation, the lips are always parted, and they sleep with the mouth open (mouth-breathers).

The third class comprises the extreme cases of adenoids. The nasal passages are the seat of a chronic hypertrophic rhinitis, the tonsils are enlarged, there is obstructed breathing, and the mouth is always open. The infants and children make a peculiar snarling sound in breathing and have a stupid look. They are not necessarily lymphatic. Many children suffering from adenoids are slightly deaf, and all are subject to repeated catarrhal attacks.

Between the extremes are seen all gradations of the affection. Many children who suffer from adenoids are well developed and in other respects perfectly normal. The deformities of the chest which have been ascribed to adenoids can hardly be so regarded. They are coincidental. Many of them are due to rachitis in early life and to unhygienic living. To trace enuresis, chorea, and masturbation to the presence of adenoids, seems also somewhat extreme. Adenoids are an obstruction to the breathing, a menace to the hearing, and also a focus for repeated infections of the nasopharynx or the ears. These are sufficient reasons for their removal.

The **diagnosis** of the condition is made from the above symptoms, and also by digital exploration. Care should be taken that the finger used in exploring the posterior nasopharyngeal space is very clean and that the nail is smoothly trimmed. The parts should not be traumatized unnecessarily. The index finger passed up and behind the soft palate encounters soft masses of adenoid tissue which bleed easily and are readily crushed. They are sometimes pedunculated, and may be attached to the roof of the nasopharyngeal space or to the posterior portion of the nares. Some authors have advised the use of a shield in exploring this space. The skilful laryngologist prefers to use the mirror in examining these parts, and protests against the digital method.

The **treatment** of adenoid vegetations belongs to the special field of the nose and throat.

Contraindications to Operations.—The tonsils and adenoids being portals of infection, there are certain states in which operations in this region may be followed by reinfection. Thus cases of chorea with endocarditis, if still active, should not be subjected to operation. The chorea is likely to recur with greater severity, and the danger of a renewed heart lesion is great. Children who are in the active stages of endocarditis or recently recovered should not be operated upon. In all these cases palliative measures, such as sprays and douches, should be employed until the conditions above mentioned are thoroughly quiescent. In one case of chorea I saw an operation for adenoids followed in three days by a chill and high fever, endopericarditis, chorea insaniens, and death within ten days. While such cases are exceptional, they teach the necessity of caution in deciding to operate upon the adenoids in chorea and heart cases.

Acute Retropharyngeal Abscess.

(*Idiopathic Retropharyngeal Abscess; Retropharyngeal Lymphadenitis.*)

The retropharyngeal space, according to Gillette, is the seat of several lymph-nodes, which are intimately connected with the lymph-vessels and lymph-spaces of the tonsils, and also with the system of lymph-vessels of the soft palate, these being also connected with the deep lymph-nodes of the face and neck. Processes such as catarrhal angina, diphtheria, scarlet fever, measles, or any lesion of the mouth, are likely to involve the retropharyngeal nodes (Karewski). Sometimes only the lymph-nodes in the median line of the retropharynx opposite the base of the tongue are affected. In this form the tumor in the midline is seen when the mouth is opened. In other cases several lymph-nodes are involved, and the process is then seen both as a swelling in the mouth and as an external swelling at the side of the neck.

The swelling appears at or beneath the angle of the jaw, in front of or behind the sternomastoid muscles. Retropharyngeal abscess may occur in the following forms:

1. Acute retropharyngeal abscess:
    - *a.* That which points wholly in the mouth.
    - *b.* That which points both externally and internally.
    - *c.* That which forms a tumor chiefly external.
2. Chronic tuberculous retropharyngeal abscess.
3. Septic retropharyngeal abscess.

This third class of retropharyngeal abscesses are those which complicate or follow the exanthemata, and which have a tendency to burrow downward, bursting into the mediastinum or to involve important structures, such as the large arteries in the neck, thus causing fatal hemorrhage. A few such cases occur in the literature.

Frequency and Etiology.—Retropharyngeal abscess is peculiarly a disease of infancy and early childhood. The frequency diminishes in later childhood, the disease being rare after the fifth year. Of 77 of my cases, 4 occurred between the first to the third month; 10 between the third and the sixth month; 41 between the sixth and the twelfth month; 19 between the first and the fifth year, and the remainder after the fifth year. One infant was only one month of age, and in two cases the patient was two months of age. The figures correspond to those of Bokai. The frequency in early infancy is probably explained by the structure of the retropharyngeal lymph-spaces and the susceptibility of the lymph-nodes to suppurative infections at that period of life.

Simon has described the lymphatics in the retropharyngeal region of infants and children as forming a small network of lymph-vessels and nodes on either side of the median line. This lymphatic network is situated between the superior constrictor and the aponeurosis of the prevertebral muscles. After the third year of life these lymphatics and nodes are said to disappear. This fact, as Blackader points out, would indicate a close connection between the time of activity of these nodes and the period when retropharyngeal abscess is most prevalent. It would help also to explain the absence of this form of abscess in older children and in adults who are frequently affected by tonsillar (quinsy) abscess.

I have examined the pus from many of these abscesses, and found that it contains quite uniformly a streptococcus of the short or the long variety, not as a rule very virulent. It may be assumed that in all probability these bacteria are the essential cause of the abscesses. They gain access to the retropharynx either through the tonsils or the mucous membrane of the pharyngeal space. The abscess may thus be secondary to any form of inflammation of these structures. It occurs as a complication of simple tonsillitis, pharyngitis, influenza, or any of the exanthemata.

The **symptoms** of retropharyngeal abscess are not at first distinctive. The development of the abscess is insidious. At the outset there are the symptoms of ordinary tonsillitis or pharyngitis. The fever is high at the beginning. After the acute symptoms subside it is noticed that the lymph-nodes at the angle of the jaw continue to be enlarged, and that the fever continues to show a remittent type. There is some prostration, the infant does not nurse properly, cries, and is frequently restless. Inspection of the throat on the fourth or fifth day of a tonsillitis may reveal nothing except some swelling or œdema of the posterior pharyngeal wall or of the pillars of the fauces, no tumor being visible. After an interval of a few days, generally on the seventh or eighth after the initial symptoms, it is noticed that the voice of the infant has a nasal quality, that the head is thrown back, and that the breathing is noisy and nasal. Examination shows that the lymph-nodes at the angle of the jaw in front or behind the sterno-mastoid are swollen; inspection of the interior of the fauces shows a distinct swelling at the side of the pharynx pushing the tonsil and pillar of the fauces of that side forward. On introducing the finger a tense, fluctuating swelling, which may reach downward toward the larynx, can be felt. In other cases there is very little external swelling, and the internal tumor is situated nearer the median line, pushing the posterior pharyngeal wall forward. This swelling is covered by mucous membrane, is tense and fluctuating. If the tumor is allowed to increase in size, there is pronounced interference with the breathing. I have seen cases in rachitic infants in which the inspiratory sound was distinctly of a crowing character, showing incoördinate action of the vocal cords. These cases show great prostration and feebleness of pulse.

Course.—If not treated, the abscess may press on the larynx and cause asphyxia, or may burst spontaneously into the larynx, suffocating the patient if it occurs during sleep, or may burst into the ear through the Eustachian tube and discharge externally. All of these results are rare if the abscess is detected in time for incision.

The **diagnosis** of retropharyngeal abscess is difficult to the beginner, but is simple after the observation of one or two cases. The quality of the voice and the cry are so characteristic that after being once heard they are unmistakable. The breathing also is typical. The external swelling is present in most cases, and the head slightly retracted. Finally, digital examination should always be resorted to in all cases in which a slight or marked internal swelling is present. The index finger of the right hand is passed into the mouth and the posterior pharyngeal wall palpated. If an abscess be present, it will be apparent as a hard or tense, globular, deep or superficially fluctuating tumor. Care should be taken not to mistake the prominence of the body of the seventh cervical vertebræ for an abscess.

The bony tumor is deeper, as a rule, than the retropharyngeal abscess, and is not fluctuating. All manipulation should be carried out gently, else the abscess may burst and suffocate the patient or rude exploration may cause a peculiar form of collapse which sometimes follows digital examination in this region.

The **prognosis** of simple acute retropharyngeal abscess is good. Bokai lost only 4 per cent. of his cases. With early diagnosis and proper treatment recovery is the rule.

The **treatment** of acute retropharyngeal abscess is incision. This varies with the nature and location of the abscess. In the majority of cases the abscess is near the median line, and its wall is just beneath the surface of the mucous membrane. An internal incision will then afford immediate and permanent relief. In other cases the abscess is at one side and internal, and may also be safely incised from within. In making an internal incision the following method should be pursued: the child is wrapped in a blanket and held upright in the lap of the nurse, facing a good light. An assistant steadies the head from behind. The tongue is depressed with a tongue-depressor, and a bistoury, with the edge guarded by rubber plaster, leaving only a half inch of the tip exposed, is plunged into the most prominent part of the tumor. When the pus escapes, the incision is enlarged from above downward. The instrument should not be directed toward the side of the neck, for fear of wounding a vessel. As soon as the pus escapes freely the head of the infant is thrown forward and the pus allowed to drain into a basin, pressure being made from without, on the side of the neck. The internal incision should be made as rapidly and as gently as possible. I have seen death result within a few hours from aspiration of pus in a case in which an abscess burst as a consequence of rough digital exploration. If necessary, the incision may be enlarged with a dressing-forceps. In some cases the wound should be prevented from closing by introducing the forceps daily.

There is another class of cases in which the deep cervical glands at the side of the neck are involved and the abscess points partly internally and partly externally. In these cases it is unsafe to incise from within, nor is complete relief afforded by so doing. The abscess should be approached from without through a careful dissection by a skilled surgeon. The tuberculous abscess is due to spinal caries, and is best opened and drained from without, as are also septic abscesses.

II. DISEASES OF THE TONSILS.

The tonsils are really lymph-nodes, as has been shown by Stöhr and Hodenpyle. In severe forms of inflammation they are enlarged, and the so-called crypts become plugged with bacteria and the products of inflammation (leucocytes, fibrin, serum). The crypts appear at the surface of the tonsil as yellowish specks. A catarrhally inflamed tonsil may not show them at the surface, because the products of inflammation do not coagulate, and are thus thrown off more readily. There is nothing specific about a lacunar or follicular amygdalitis. It is only a clinical picture of the large class of catarrhal inflammations, in all of which the crypts and the tissue of the tonsil are infiltrated with inflammatory products.

Acute Follicular Amygdalitis.

(*Acute Catarrhal Tonsillitis; Acute Lacunar Amygdalitis; Catarrhal Angina.*)

Acute follicular amygdalitis is an infectious disease, communicable either through the secretions or by direct contact, as in the act of kissing. It occurs both as a primary and as a secondary affection. As a primary affection, it occurs at all periods of infancy and childhood. It was formerly taught that follicular amygdalitis was rare in infants. This is scarcely true. Of 1284 cases of lacunar amygdalitis, 333 occurred in infants under the age of twelve months, and 76 from the first to the fifth month; of the latter, only 5 occurred in the first month. It is frequent in children from the second to the fourth year, but is more common after than before the fourth year. The tonsils are secondarily involved in the exanthemata—scarlet fever, measles, and varicella—and in parotitis, influenza, pneumonia, and pertussis. In all these affections they are red, swollen, and in some cases present the appearance seen in the typical lacunar type of the disease.

Etiology.—The predisposing causes of catarrhal tonsillitis or lacunar amygdalitis are exposure to cold, traumatism, and the swallowing of corrosive or irritant substances. The exciting causes of follicular or lacunar amygdalitis and catarrhal amygdalitis are the Streptococcus pyogenes, the Staphylococcus pyogenes, and the pneumococcus. The diplococcus described by Roux is also found in the tonsillar crypts.

Symptoms.—The affection rarely begins with a chill. The infant is restless, peevish, and wakeful at night; it breathes rapidly, and there are high fever and marked prostration. Nursing is interfered with, not only on account of the pain in swallowing, but because in the majority of cases there is more or less rhinitis present. As a rule, the bowels are disturbed as a result of swallowing infectious secretions from the mouth with the food. The action of the bacteria in the gut is manifested in green stools, which are frequent and watery. Inspection of the throat should be conducted with patience and in a good light. The tonsils, normally very small, are seen to be enlarged and studded with whitish or yellowish-white points. The lymph-nodes at the angle of the jaw may be enlarged.

In older infants and children the tonsils are enlarged, and the crypts plugged with inflammatory products. The surface of the tonsils is covered with mucopurulent exudate, or there may be a small necrotic, ulcerated area in one of the tonsils. The neighboring structures, such as the uvula, the pharyngeal mucous membrane, the pillars of the fauces, and even the larynx, may share in the catarrhal inflammation. The lymph-nodes at the angle of the jaw may be enlarged. The fever, as a rule, is high at first, ranging from 104° to 105° F. (40° to 40.5° C.) or above. The pulse is correspondingly rapid, and the respirations may be increased in frequency.

The duration of a typical case of primary tonsillitis varies. As a rule, the temperature remains high for two or three days, with daily remissions. It then subsides and the patient convalesces. In some cases the temperature continues high for five or ten days, and then drops. In all of these cases there is some latent or apparent complication, such as retropharyngeal abscess, otitis, or, as has been recently pointed out by Packard and others, an insidious endocarditis. When otitis supervenes the tonsillar affection subsides. The fever, however, continues, with daily remissions. As a rule, infants and young children do not indicate the existence of pain in the ear. The patient is restless at night, and wakes with a start or in a peevish mood. In many cases the otitis can be diagnosed only by exclusion. In other cases the temperature continues high for a week or longer, reaching 103.5° F. (39.7° C.) during the day. The infant seems weaker, the tonsils are not enlarged or severely inflamed, the pulse is accelerated, and the respirations may number 40. In such cases the lungs show no sign of involvement, but careful examination of the heart will often reveal the presence of a systolic murmur at the apex and a slight increase of the area of cardiac dulness beyond the nipple. These are the so-called rheumatic cases. Frequently the urine shows a trace of albumin. In rare cases it contains in addition to the albumin elements pointing to parenchymatous irritation of the kidney. I saw a case recently, in a child six years of age, in which after a mild attack of tonsillitis there were a few casts, blood-cells, and a small amount of albumin in the urine. Months elapsed before the urine ceased to show evidences of the nephritis. In these cases the albuminuria may assume the so-called cyclic character.

The **prognosis** of simple catarrhal tonsillitis is good, recovery taking place in a few days. On the other hand, tonsillitis is not the simple entity formerly supposed. In infants and children this is especially true. The physician should be watchful for possible complications and sequelæ, such as otitis, retropharyngeal abscess, endocarditis, and nephritis.

The **diagnosis** of tonsillitis is usually a simple matter. If an infant refuses the breast and the temperature is elevated, the throat should be carefully inspected. It is good practice to make a bacteriological culture with the secretions from the throat, even though the appearances are not diphtheritic at the first visit (for technique, see section on Diphtheria).

The **treatment** of acute tonsillitis is symptomatic. Sponging with cold water or water at 85° F. (29.4° C.) containing a dash of alcohol, will lower the temperature. A dose of quinine should be given twice daily, and if the lymph-nodes at the angle of the jaw are enlarged, cold applications should be made externally. Sprays are not required unless there is a harassing cough. Dobell's solution sprayed three times daily will relieve that symptom. In nursing infants the number of feedings by the breast or bottle is reduced. If there is disturbance of the bowel, a teaspoonful of castor oil or grain ½ (0.03) of calomel, given twice daily, will empty the bowel. The infant is then dieted on albumin-water or barley-water, or a solution of acorn cocoa or beef-juice and barley-water, until the intestinal irritation has disappeared. A return to a milk diet may be made as soon as the movements become normal. Small doses of ferric chloride have a beneficial effect on older children. In mixture form it is an excellent local application to the tonsils. The custom of giving potassium chlorate in this mixture is now generally abandoned, the drug being highly irritant to the kidneys. In nursing infants ferric chloride causes diarrhœa. For this reason it should not be administered to them for long periods.

Herpes of the Tonsils.

Herpes of the tonsils are small vesicular formations seen on the anterior pillars of the fauces, just in front of the tonsils. They occur in a number of slight febrile conditions, may accompany an angina of a simple type, and are part of the clinical picture of aphthous stomatitis. The vesicles burst, leaving yellowish ulcerations of the size of a pin's head and surrounded by a pink areola. They heal without treatment after a few days.

Ulceromembranous Tonsillitis or Angina.

(*Associated with the so-called Fusiform Bacillus of Vincent.*)

This is a peculiar affection occurring in children. At first one tonsil is affected, generally the right. After a few days the affection may spread to the other tonsil. Most of the cases I have seen were unilateral. In addition to the tonsillar ulcerations, a stomatitis of an ulcerative type is often present, and there may be ulcers on the tongue, cheeks, and gums.

The size of the tonsillar ulcer varies from that of a lentil to an involvement of a greater part of the tonsil, the shape of the ulceration being irregular, and its character rather of a chancroidal type. It has a worm-eaten base with sharp, overhanging edges, which may be slightly raised above the surface of the tonsil. The rest of the tonsil is but very slightly inflamed. The color of the ulceration is a yellowish-green gray, or dirty brown, and from the first it appears as though the base of the ulcer were covered by membrane. The depth of the ulcer is quite considerable, varying from $\frac{1}{8}$ to $\frac{1}{2}$ inch. The submaxillary glands may be enlarged, or the lymph-nodes communicating with the tonsil at the angle of the jaw may also be enlarged.

Etiology.—The etiology of ulceromembranous tonsillitis or angina has been carefully worked out by Frühwald, Vincent, Lemoine, Abel, and in our own country by Sobel, Herrman, and others. This form of tonsillitis is caused by a bacillus, described more particularly by Vincent, and a spirillum. The bacillus is about twice as long as the diphtheria bacillus, is pointed at both ends; in other words, fusiform. Some of the bacilli are bent into crescent shapes. They vary in size, some being larger and thicker than others. The spirilla are long, corkscrew-like, with wide curves. They also vary in size, the larger and thicker ones staining more deeply. The bacilli and spirilla are motile.

Symptoms.—This affection can scarcely be classed as one of the more serious affections of the tonsil, although at times of a subacute chronicity. The children are brought to the physician with a history of an ordinary sore throat, and when examined this ulcer of a deep-spread, pseudomembranous type is found on one or the other tonsil. The appearance is as if an irregular hole were punched out of the tissue of the tonsil. There is no spreading of membrane, nothing resembling diphtheria. There is slight fever, rarely higher than 103° or 105° F. The symptoms at the outset are so mild that when the patient is brought to the physician the ulceration has taken place. In those cases in which there is accompanying stomatitis on the tongue, gums, or buccal mucous membrane, there is also fœtor of the breath. In some cases there may be pallor of a distinctly septic type.

Diagnosis.—The clinical diagnosis must be made from that of diphtheritic ulcers, resembling very much what has just been described. Henoch and the author have described ulcers of a truly diphtheritic character very much resembling ulceromembranous angina. The only test is that of the culture-tube or the smear. An ordinary microscopical smear stained from the base of the tonsillar ulcer will reveal its true character if of the Vincent type. If the bacillus and spirilla are not evident at once, we should make a culture for the diphtheria bacillus.

The **prognosis** is invariably good; although in some cases the course of the disease is apt to become subacute, on account of the difficulty of reaching the base of the ulcer with remedies. Some cases may last as long as three weeks; others recover within a few days. Lemoine relates one case which lasted seventy days.

The **treatment** is much the same as that of an ordinary tonsillitis. The tincture of the chloride of iron is given in doses of from 3 to 5 minims, combined with glycerin and water, every three hours. The base of the ulcer may be touched daily either with Lugol's solution or a 10 per cent. solution of nitrate of silver.

III. DISEASES OF THE LARYNX.

Acute Catarrhal Laryngitis.

(*Catarrhal Croup; Spasmodic Croup; Spasmodic Laryngitis; Pseudocroup.*)

Etiology.—Exposure to cold or wet are predisposing causes. Like the majority of catarrhal inflammations of the respiratory passages, acute catarrhal laryngitis is due to the invasion of bacteria. It occurs as a primary affection, and in a modified form is met with secondarily in measles and influenza. The classical form of "croup" is a primary affection, and is most common from the second to the fifth year. It is also seen in very young infants. One attack predisposes to others.

Symptoms.—Catarrhal croup or catarrhal laryngitis is an affection that causes much concern to mothers when a first attack develops without warning. During the day the infant may have had a mild coryza with a slight elevation of temperature. Toward evening a croupy cough, accompanied by croupy breathing or voice, suddenly develops. In some cases the symptoms remain mild, and only the cough disturbs the patients. They breathe freely, and dyspnœa is not marked. In other cases the infant or child goes to sleep free from alarming symptoms. Coryza may have been present unnoticed during the day. During the night the patient awakes with a croupy cough, which rapidly becomes worse. The breathing is noisy (croupy), and may be heard in an adjoining room. The cough is especially terrifying. The patients are restless, and cry during the paroxysms of coughing. In some cases they sit upright and gasp for breath. The face is pale and wet with cold perspiration. Fever may be slight or marked and may reach 104° F. In the majority of cases the dyspnœa is real; there is drawing inward of the suprasternal region and the peri-pneumonic groove at the epigastrium. Toward morning the dyspnœa, cough, and croupy breathing subside, and the patients fall asleep, worn out with the night's suffering. The next day the patients are apparently well, with the exception of a slight or marked croupy cough, coryza, swollen tonsils, with redness of the pharynx. For two or three successive nights or days there may be a repetition of the attack. This condition should be differentiated from laryngismus stridulus. In the latter there is no fever, the breathing is stridulous during only a short spasmodic attack, and there is no croupy cough. On the other hand, pseudocroup may occur in children who are rachitic and the subjects of laryngismus. There are forms of diphtheritic laryngitis without the formation of membrane, which in their symptomatology are identical with the form of laryngitis above described. This is true in very young infants and in children above five years of age. A culture-test is the only certain mode of differentiating the affections. The pathological condition giving rise to pseudocroup is believed to be a swelling of the mucous membrane beneath the vocal cords.

Diagnosis.—The diagnosis is not difficult except in cases in which the croupy cough, breathing, and stridor increase as the day or night wanes and no relief comes to the sufferer. In other cases the obstruction to the breathing in the larynx increases as in truly membranous cases. Only a *repeated* culture will reveal the nature of such an affection, because one culture may be negative even in a truly diphtheritic case. In the severe forms of "croup," in the face of increasing laryngeal obstruction, the interests of the patient are best subserved by assuming the presence of a diphtheritic process until the bacteriological culture proves the contrary to be the case.

The **prognosis** is good. I have never met a fatal case of non-diphtheritic catarrhal croup. On the other hand, many of these cases are due to a grippal infection. Such an infection may carry in its train complications, such as bronchopneumonia or ear affections, which may endanger the life of the patient.

Treatment.—The patient is isolated, and placed under a tent improvised over the crib. The tent is kept filled with steam generated by any of the devices for croup in the market (croup-kettle); the steam is saturated with turpentine, thymol, or benzoin. At intervals of an hour 10 grains of calomel are sublimed underneath the tent until the croupy cough and breathing abate. To relieve the laryngeal spasm, especially if there is a temperature, antipyrin, in doses of a grain to every year of the age, is efficient and induces rest and sleep. Antimony ($\frac{1}{100}$ grain) combined with ipecacuanha ($\frac{1}{100}$ grain) may be given every two hours, or 20 drops of the syrup of ipecac every two hours until emesis occurs. Turpeth mineral is given by some to induce vomiting. I do not use the drug. If symptoms of progressive stenosis set in, intubation is justified, and in localities where bacteriological examinations are not feasible, diphtheria antitoxin should be administered, lest a membranous diphtheritic process be overlooked. I have seen cases, however, which developed cyanosis recover without intubation. It is questionable whether it is justifiable to allow the patient to suffer when such a simple means, as the introduction of a tube in the larynx, is feasible. The application of counterirritants to the larynx is of questionable utility. The same may be said of the application of heat or cold externally.

Œdema Glottidis.

(*Submucous Laryngitis; Phlegmonous Laryngitis.*)

Definition.—This is a serous or sero-purulent infiltration of the submucous cellular tissue of the region of the upper larynx, or glottis, and the aryepiglottic folds.

Etiology.—There are two forms—first, the simple serous infiltration of the glottis; and, second, the inflammatory infiltration, the so-called phlegmonous laryngitis, in which the submucous connective tissue is involved. The serous form is secondary to and accompanies acute and chronic nephritis, infectious diseases, scarlet fever, variola, syphilis, typhoid fever, inflammation or ulceration of the structures adjacent to the larynx, especially of an erysipalatous nature.

The second form, the phlegmonous laryngitis, is due to traumatism, such as the direct inhalation of steam, customary among children of the tenements when playing in the kitchen; chemicals, foreign bodies, and injuries.

Morbid Anatomy.—In the serous form of œdema glottidis the submucous tissue is tense, infiltrated, pale or yellowish red; there is swelling of the upper laryngeal area. In the phlegmonous form the mucous membrane is dark red, swollen, covered with pus, and there may be ulceration of the mucous membrane of the larynx and vocal cords.

Symptoms.—In the forms accompanying nephritis and the infectious diseases, the first symptoms to appear are those of stenosis of the larynx. In the traumatic form of phlegmonous laryngitis with consequent œdema of the glottis, especially in cases in which steam has been inhaled by children, there is pain in the mouth and pharynx, dysphagia, and dyspnœic attacks. Inspection shows the mucous membrane of the mouth and pharynx to be inflamed and the tissues of the epiglottis swollen; and an inspection of the larynx reveals swelling of the false vocal cords and narrowing of the rima glottidis.

The **course** of the disease depends on the nature of the primary affection. The milder cases, especially those accompanying acute or chronic nephritis, may retrograde. Other cases, especially the traumatic, if unrelieved may result in fatal suffocation.

The **prognosis** must depend on the prognosis in the first form of the primary affection, and in the traumatic and phlegmonous forms of laryngitis the prognosis of the œdema glottidis depends on the severity of the disease.

The **treatment** must consist, if a nephritis be present, in the treatment of the nephritis, and we must not forget that intubation or scarification in children is in most cases ineffectual. Intubation is apt to be ineffectual on account of the additional traumatism caused by the attempts at introduction of a tube, with consequent formation of false pockets. If the symptoms are such that suffocation is imminent, tracheotomy offers the simplest and safest means of relief.

In many cases of œdema of the glottis, especially of the milder type, a small dose of the opiates will quiet the patient and have a tendency to relieve the apparent dyspnœa until such time as the symptoms of the primary disease retrograde. Especially difficult of treatment will be the secondary cases, with phlegmonous disease involving structures adjacent the larynx, such as angina Ludovici. In these cases the swelling of the structures may be so great as to make tracheotomy a very difficult operation. Intubation in these cases is scarcely to be thought of.

Syphilis of the Larynx.

This affection is rare in infancy and childhood, inasmuch as it accompanies the later forms of syphilis. The seat of election of this disease is the epiglottis, where ulcers and condylomata are formed. The structures are thickened, inflamed, covered with white, diffuse patches, and the same changes are seen in the interior of the larynx as on the aryepiglottic folds. Cicatrices may form and cause marked symptoms of stenosis. Ulcers are seen on the back of the tongue and on the vocal cords. Gummatous infiltrations may form, ulcerate, and lead to inflammation of the cartilages and necrosis of these structures, causing stenotic symptoms.

The **diagnosis** depends on a discovery of syphilitic lesions elsewhere.

The **prognosis** depends on how soon anti-syphilitic treatment can be in augurated before ulceration and cicatrization results.

The **treatment** of this affection consists in applying the anti-syphilitic remedies; and when stenosis of the larynx occurs as a result of cicatrization and contraction of the structures of the larynx, intubation, according to O'Dwyer, offers the most effective means of relief.

Tuberculosis of the Larynx.

This is very rare in infancy and childhood, and is more common toward the age of puberty. It can affect any part of the larynx, causing hoarseness. It is rarely primary, being, as a rule, secondary to tuberculosis of the lungs or other organs.

The **treatment** belongs in the realm of special laryngeal work.

Growths in the Larynx.

The most common tumors found in the larynx are papillomata, granulomata, and fibromata. Malignant tumors are rare. Fully 25 per cent. of the papillomata are congenital, and manifest themselves from birth by symptoms of hoarseness and troubled cough. A frequent case of granulomata and papillomata of the larynx is recurrent laryngitis and operations upon the larynx, such as intubation or tracheotomy.

Symptoms.—Tumors of all kinds cause hoarseness, accompanied by paroxysms of coughing with difficulty of respiration due to a certain amount of stenosis, varying according to the size of the tumor. Some of these tumors may give rise to symptoms of suffocation. The granulomata which follow tracheotomy cause symptoms of asphyxia after the removal of the tube.

In addition to the above symptoms, there are evidences, in all cases of tumor of the larynx, of catarrhal inflammation of the neighboring structures.

The **treatment** of growths in the larynx belongs to the **realm** of throat surgery.

Foreign Bodies in the Larynx.

During play children often aspirate bodies of all kinds into the larynx, and the symptoms caused depend very much upon the size and shape of the body aspirated. In rare cases the body lodges in the larynx, and may cause instant death by suffocation. Smaller bodies lodging in the ventricle of the larynx may cause attacks of dyspnœa, which subside when the patient takes the recumbent position; but even these small bodies may cause instant death if they once lodge in the rima glottidis and close the opening of the larynx. Some of these bodies may after a time lodge in the bronchi and cause pneumonia.

The **prognosis** depends upon the nature of the body and the possibility of dislodging it.

Treatment.—If the body is small, it may sometimes be dislodged by standing the patient, as it were, on the head. It then emerges into the larynx and is coughed out. If such is not possible, it is best to locate the body by means of a radiograph, and then attempt its removal by surgical means.

IV. DISEASES OF THE BRONCHI.

Acute Simple Bronchitis.

Bronchitis, acute and simple, is an affection of the larger and medium-sized bronchi. In very young infants the disease is apt to be very severe and to attack the smallest bronchioles; it is then called capillary bronchitis. A capillary bronchitis is really a bronchitis in which there is a certain amount of peribronchitic pneumonia. Acute bronchitis may occur at any period of infancy or childhood. It is, however, less common before the sixth month of infancy than during the period up to the third year, when its frequency diminishes.

Causation.—Bronchitis may be caused by an exposure to cold or wet or by traumatism to the mucous membrane of the air-passages through the inhalation of dust or irritating vapors. It occurs in the acute infectious diseases, such as malaria, scarlet fever, measles, rötheln, varicella, typhus and typhoid fevers, and frequently complicates pneumonia of the lobular or lobar type. Rachitis and syphilis predispose to attacks of bronchitis. The bronchitis of heart disease or nephritis should be regarded as of a different class.

Pathology.—The bronchi may be filled with a mucous, serous, purulent, or mucopurulent secretion, which is secreted by the epithelium of the mucous membrane and the mucous glands in the wall of the bronchi. In recent acute bronchitis the mucus is quite abundant. In the exudate on the mucous membrane of the bronchi and in the lumen, epithelial cells, leucocytes, and sometimes red blood-cells are found. The structure of the mucous membrane is infiltrated with small round cells to a greater or less degree. In some places the epithelial lining of the bronchi may be raised by exudate; in others there may be loss of the superficial epithelium. If the bronchitis lasts any length of time, there may be atrophy of the structures of the mucous membrane. In the severer forms of bronchitis which affect the smaller bronchi the peribronchitic connective tissue is infiltrated with small round cells. In these cases there is an inflammatory exudate in the surrounding alveoli of the lung. There is then peribronchitis or bronchopneumonia.

Symptoms.—In some cases the infant or child suffering from acute bronchitis will have a simple angina as an initial symptom. There is mild redness of the fauces with a slight rise of temperature which may last a day or more. The cough which was present at first persists, and there may be slight disturbance of the bowels, the movements being green and containing large curds of undigested matter.

The cough may in aggravated cases give rise to occasional attacks of vomiting, especially immediately after nursing; at other times the coughing spells may cause the patient to cry. There is evidently pain, especially in the cases of bronchitis affecting the larger bronchi. The infant sometimes suffers from great difficulty in expelling the accumulated secretion. The attacks of coughing closely resemble those seen in old people who suffer from bronchitis. In many cases the infant or child is quite comfortable in the intervals between the coughing spells. In others the respirations are increased, and there may for some days be a slight evening rise of temperature, the patient showing signs of being seriously ill. In very young infants who are rachitic there may be a distinct drawing in of the sides of the chest and of the peripneumonic groove at each respiration. In cases of severe involvement of the smaller bronchi, there may be slight cyanosis of the lips and pallor of the surface.

In the severer forms of bronchitis, especially of the grippal variety, there is a distinct rise of temperature for several days. It may rise to 102°–103° F. (38.8°–39.4° C.), or even higher, with a corresponding increase in the number of respirations and the pulse-rate. In weak and very young infants there may be little or no cough. The infant lies in a soporose state, does not nurse well or refuses the breast. Older children may run about and play while suffering from bronchial trouble; severe bronchial disturbance may appear to have little effect on the general health. Expectoration is very exceptional; a frothy mucus collects about the lips of young infants after an attack of coughing. In older children it may be very difficult to collect sputum, even if they are old enough to understand the necessity of expectorating the secretion. The conclusion has been that children swallow the expectoration; it is more rational to suppose that the efforts at coughing are not equal to raising any considerable quantity of secretion or that the amount of secretion in bronchitis is not so great as has been generally supposed. In many cases the cough is severer at night than during the day, but children cough and fall asleep immediately afterward, and therefore do not lose much rest. I have never met with a simple acute bronchitis ushered in by a chill or convulsion. I have, however, seen severe forms of bronchitis cause petechial extravasations on the skin, similar to those seen in pertussis. The petechiæ are apt to occur about the forehead and eyes of very weak infants.

Physical Signs.—In mild cases the respirations may be slightly above the normal; in severer cases there are signs of dyspnœa and the respirations are increased in number. In very severe forms the peripneumonic groove may be drawn inward with each respiratory act. In capillary bronchitis the lips may show some cyanosis, the surface may be pale, and the finger-tips slightly cyanosed.

Palpation.—If the palms of the hands are placed in front and behind the chest, the so-called rhonchal fremitus may be elicited. The vibrations caused by accumulated secretion in the large and small bronchi give a sensation resembling that felt in stroking a purring cat.

Percussion.—In simple acute bronchitis, percussion may elicit nothing abnormal. If infants have suffered from repeated attacks of bronchitis, the note may, owing to a slight emphysema, be hyper-resonant or vesiculotympanitic. In severe forms of capillary bronchitis there may be areas of peribronchitic pneumonia or bronchopneumonia, over which careful percussion will detect slight dulness with a resonant note.

Auscultation.—In a vast number of cases, bronchitis at the outset, gives on auscultation nothing but a rude respiratory murmur which is more markedly puerile than is normal. As the secretion accumulates there will be sonorous, sibilant, and subcrepitant râles, and also sonorous breathing. In the form called capillary bronchitis, with the subcrepitant râles there will be râles of much finer quality, resembling crepitant râles. The latter, which are unmistakable, are heard on inspiration, and appear to indicate areas of peribronchitic pneumonia. In newly born and weakly infants there are, in this form of bronchitis, areas in which the air is not heard to enter the lungs (atelectasis).

The **treatment** of simple acute bronchitis should be supporting and expectant. If the cough is harassing, a mild opiate mixture in combination with a small quantity of ipecac may be given. The following prescription has been found useful:

℞	Tinct. opii camph.	ʒj (4.0).
	Syr. ipecacuanhæ	♏ xxxij (2.0).
	Syr. tolutani	℥ij (60.0).

Sig. Teaspoonful every three hours.

The patients are allowed to be in the open air in fine weather, and the room should be well ventilated at night. In cases in which there is great relaxation of the mucous membranes, a dose of strychninæ sulph., grain $\frac{1}{200}$ (0.0003), may be given three or four times daily. The child is kept warmly clad, and wool is worn next the skin. Douching with cold water is to be avoided in acute cases. The oil-silk jacket may be worn, but it has no special superiority to warm clothing. Applications of oil to the chest are of no value. The drugs of the coal-tar series (antipyrin or phenacetin) should not be used, except that one dose may be given at the very outset to relieve restlessness or headache. The bowels should be relieved by means of calomel or a saline cathartic.

In the subacute stage, syrup of ferric iodide may be given as a tonic for the mucous membrane. In very rachitic infants and children, cod-liver oil is indicated.

The treatment of so-called capillary bronchitis approaches very closely that of bronchopneumonia. The heart should be supported. Digitalis in the form of tincture is the most useful remedy. Strychnine, caffeine, camphor, and musk in form of powder, all have here their legitimate sphere.

The temperature, as a rule, needs no treatment. With older children, if the secretion is very profuse, carbonate of guaiacol is exceedingly useful and gives much relief.

Fibrinous or Plastic Bronchitis.

This is a form of bronchitis in which membranous masses or fibrinous exudate are coughed up at intervals. These masses may have the exact shape of the bronchi, or may consist of shreds or bands of membrane.

Etiology.—Bronchitis of this form complicates diphtheria and pneumonia, and also occurs in the acute infectious diseases—measles, scarlet fever, tuberculosis, erysipelas, typhus and typhoid fevers. It is found in diseases of the heart and lungs, and may result from traumatism through the inhalation of poisonous gases. The above are the secondary forms; the primary form of fibrinous bronchitis is obscure in its etiology, and is rare in infancy and childhood.

Morbid Anatomy.—The casts which are coughed up are cylindrical in shape and branched in the form of the larger and smaller bronchi. The larger ones may be hollow and cylindrical, while the smaller ramifications may be solid or thready. In other cases the whole cast is solid; small air-bubbles may be confined in the fibrinous cylinders. The casts may be 10–12 cm. in length, the extremities being nodular, thready, or flat. Under the microscope the casts are seen to be formed in layers; in the centre of the oldest layers are found epithelium of the bronchi, leucocytes, and bacteria. Spirals formed of fibrin are occasionally found in the expectorated masses, especially in the diphtheritic, pneumonic, and the so-called idiopathic cases.

Symptoms.—**Attacks of Dyspnœa.**—This form of bronchitis is characterized by attacks of dyspnœa and coughing. During the attacks clots of purulent fibrinous masses are expectorated, sometimes with a slight amount of blood. In spite of the expectoration of blood there are no signs of tuberculosis. The presence of blood is probably caused by the detachment of the membranous casts from the walls of the bronchi. The expectorated masses may contain asthma crystals. In the intervals between the attacks, there may be symptoms of an ordinary bronchitis with mucopurulent expectoration, or there may be absolute freedom from symptoms.

The **cough**, which is present during the attacks, may be accompanied by a snarling or fluttering sound.

Cyanosis may be present during the attack to a marked degree or may be absent.

Fever is present in the acute form, but has no special characteristics.

Splenic tumor may be present.

The physical signs of bronchitis may be present with râles of all kinds. If the membranous masses hang detached in the bronchi, a snarling or flapping sound may be heard on auscultation.

The general condition of patients in the intervals and during the attacks varies greatly. In some cases it is fairly good.

Complications.—A tuberculous bronchitis or pneumonia may be a complicating condition.

The **diagnosis** is made from the presence of the fibrinous casts.

The **treatment** has thus far been very unsatisfactory; mercury, and also inhalations and sprays of all kinds have been tried by Biermer in the acute cases. Iodide of potassium is of value in the intervals. If diphtheria is present, the antitoxin is given.

Emphysema and Chronic Bronchitis of the Lungs.

Frequency.—Emphysema is a condition frequently seen postmortem in the lungs of infants and children (Steffen). No disease of the lungs runs its course without causing some emphysema. The condition is much more common in children than in adults, because it is favored by the peculiar structure of the lung during early life. Most of the forms of emphysema of the lungs of infants and children retrograde, allowing the lung to return to its normal state. Otherwise emphysema would be more common in adult life than it is. Clinically, emphysema combined with various forms of pulmonary disturbance, especially bronchitis, is very common in infants and children. My experience in this respect confirms that of Steffen and Osler. It seems to be common to certain classes of children, especially those of rachitic tendencies.

Morbid Anatomy.—Steffen has made a very careful study of the pathological condition in emphysema of the lungs of infants and children. The thorax has not the typical barrel shape seen in the adult, and occasionally found in older children. In younger children, especially in those with rachitis, the sides of the lower portion of the thorax are incurved; the upper part of the thorax in front underneath the clavicles may be full and prominent. On opening the chest, the lungs are found to be inflated, to retain their form, and to show along the situation of the ribs a series of indentations due to pressure. The depressed portions may be denser than those raised, and show areas of circumscribed persistent pneumonia. In vesicular emphysema, air-vesicles may rupture into one another, giving rise to large sac-like formations which communicate with a bronchus. Some of the air-vesicles may rupture into the subpleural tissue. Vesicular emphysema rarely involves a whole lung or both lungs, but is localized to certain areas, such as the apices, anterior borders, or the lingula. The emphysematous areas are whitish, yellowish white, or reddish yellow, the color varying with the amount of blood contained. They are raised above the surface, are elastic and velvety to the touch, and crepitate with the air contained. In children, in contrast to the condition in the adult, the heart is rarely dilated, and the liver and kidneys rarely affected. This is due to the temporary nature of the process. Bronchitis, trachitis, and laryngitis may exist as primary or secondary conditions. It is not possible to consider emphysema in infants and children as an isolated condition. Since it is most frequently seen in pronounced bronchial affections, it will be convenient to consider it in connection with bronchitis.

Symptoms.—Some infants and children suffer from a chronic catarrhal bronchitis which is more or less present at all times, and which may be interrupted by attacks of acute bronchitis. Infants and children thus affected are more or less rachitic; some have lymphatism in the form of chronic hypertrophic rhinitis and also adenoids or enlarged tonsils. In the intervals between the attacks of acute bronchitis, the patients do not seem to suffer much constitutional disturbance. There is no fever, and no change in the respiration except that it assumes a noisy character. There is a cough which comes on at intervals, especially at night. The infants are pale, with rather flabby muscles, and may be fat, but impress the physician as being below the normal in point of strength.

Physical Signs.—If the bronchitis has persisted a long time, the upper part of the chest is, even in infants under the age of twelve months, abnormally full. The upper costosternal region is high and the intercostal spaces are filled out. In milder cases there are no signs to be detected on inspection.

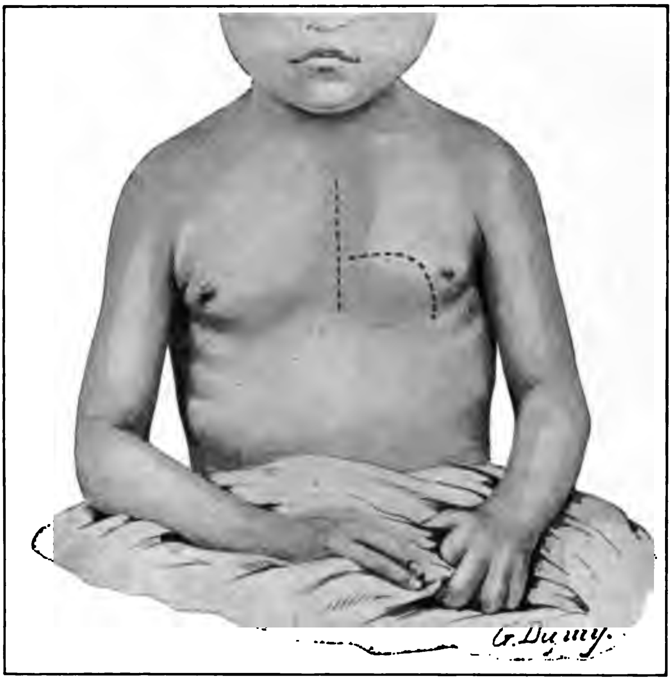
Palpation.—There is distinct rhonchal fremitus felt anteriorly and posteriorly.

Percussion.—If there have been a number of acute attacks, there will be emphysema of a vesicular type, giving a hyper-resonant note. In pronounced rachitis the hyper-resonance is apt to be marked. The area of relative cardiac dulness in older children is much diminished (Fig. 102).

Auscultation.—Voice-sounds are normal. The breathing is rude or sonorous. The respiratory murmur may be prolonged. There are sonorous, mucous, and subcrepitant râles.

A second set of cases of chronic bronchitis comprises those in which a condition of pronounced emphysema of a vesicular character is present, and in which there are distinct attacks of dyspnœa or asthma. These cases must be differentiated from the purely neurotic cases of spasmodic asthma. The latter condition is rare in children, and is not accompanied by chronic catarrhal bronchitis. The history of these cases is one of repeated attacks of acute bronchitis. The lung may in the interval be wholly free from signs of bronchitis. A condition of this kind is apt to be left in the lung after a severe attack of pertussis. The infants or children may bear the marks of rachitis, and are usually anæmic. In the intervals between the acute attacks of asthma, the general condition is good. There is no fever; there may be dyspnœa on exertion. An attack of asthma is precipitated by exposure to cold or wet. During the attacks infants and children do not suffer much, although they show signs of marked dyspnœa. There are none of the typical signs of an attack of spasmodic asthma in the adult. An infant showing very marked dyspnœa will play in the arms of the mother. The lips may be cyanosed and the surface pale and cool. There is no temperature. There is in these subjects a tendency to develop a cough of a laryngeal type on the least exposure. Examination of the chest shows nothing except a prolonged rude respiratory murmur, while percussion will give a hyper-resonant note over the whole chest. Suddenly an attack of so-called asthma will develop, with all the physical signs given below. The onset of the attack is sometimes signalized by a slight rise of temperature, 100° to 101° F. (37.7° to 38.3° C.), and an increase in the number of respirations, 32 to 36 per minute. On examination, the chest shows all the signs of an acute attack of bronchitic asthma. An attack lasts for from a few hours to a few days. The children usually play about and seem little disturbed by their condition.

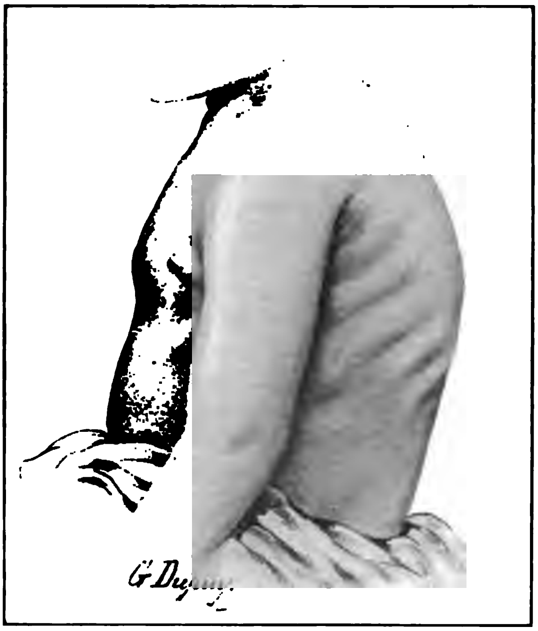
FIG. 102.



Emphysema of the lung in a boy eight years of age; diminished cardiac area of relativ dulness.

Physical Signs.—*During an attack of spasmodic dyspnœa:* **Inspection** shows a drawing inward of the supersternal structures on inspiration, and a depression of the peripneumonic groove. The upper part of the chest is high and filled out, and moves little on inspiration and expiration. The lower part of the thorax has also little movement. In rachitic children, there is not only drawing inward of the lower part of the thorax, but also a distinct incurvation of the lower ribs, caused by the repeated attacks of dyspnœa. The chest is moved as a whole. In children of seven or eight years the dyspnœa may be severe in the absence of cyanosis. These patients apparently suffer more than infants.

FIG. 103.



Emphysema of lung: boy eight years of age; barrel-shaped thorax. Same patient as Fig. 102.

In older children, the chest has the typical barrel shape seen in the adult sufferer from asthma (Fig. 103). In one case, my notes describe a drawing inward of the intercostal spaces. Some cases have a constant cough and frothy expectoration.

Palpation gives rhonchal fremitus and faint cardiac impulse.

Percussion gives a vesiculotympanitic or hyper-resonant note over the whole chest, and cardiac dulness obscured and diminished by the emphysematous lung.

Auscultation gives a prolonged expiratory murmur and sibilant and sonorous râles. Heart-sounds are feeble.

Between the attacks of dyspnœa the chest retains the above forms. There may be a slight constant dyspnœa or none at all. The patient feels quite well, and does not complain of the dyspnœa. The heart apex-impulse is diffused.

Palpation gives little or no rhonchal fremitus. Percussion shows a note hyper-resonant, but not as markedly so as during the paroxysm of dyspnœa. Cardiac relative dulness is obscured by the presence of emphysema.

Auscultation.—In older children the expiratory murmur may be prolonged or inaudible. There are signs of residual bronchitis, sibilant, sonorous, and subcrepitant râles, and in young infants, large mucous râles. The signs may be hardly noticeable or heard only in certain portions of the chest.

Prognosis.—In both forms of chronic bronchitis the prognosis quoad vitam is very good. The chances of ultimate restoration of the lung to the normal condition depend much on the mode of living and the power of the individual to outgrow the conditions of rachitis and lymphatism which exist in many of these cases. Many of these forms of chronic bronchitis disappear ultimately; the emphysematous form may persist into adult life.

The **treatment** of chronic bronchitis is directed toward improving the general tone of the economy and also the musculature of the heart. It must be assumed that in these cases the heart as well as the other organs suffers from a lack of power, to which may be attributed the relaxed condition of the circulation in the mucous membrane of the bronchi. Life in the open air, hydriatic treatment, and heart tonics, such as strychnine, will have beneficial effects. The mucous membranes are benefited by preparations of iron which contain iodine (syrup of the iodide of iron), freshly prepared and given in large doses. Cod-liver oil is an excellent tonic in winter. The skin should be protected from extremes of heat and cold by suitable underwear. Moderate participation in sports in the open air improves the action of the heart. Running and gymnastics are to be preferred to bicycle-riding.

A dry climate will do much toward improving the condition of the lung. During the attack of dyspnœa, iodide of potassium will be of service in alleviating the symptoms. This is the most useful remedy. It is also of great benefit when given in the intervals between the attacks. The other drugs used with adults are not indicated. An exception is Fowler's solution, which is an exceedingly useful remedy in moderate dosage in the intervals of the attacks, to be given, over a prolonged period. I have seen good results follow the use of digitalis in the form of the tincture, in combination with the iodide of potassium. The heart is thus greatly aided in improving the circulatory conditions in the emphysematous lung. Rest from exertion is indicated during the attack, but patients may be kept out of doors if they will remain quiet. Codeine is most useful in allaying the cough. The administration of a large dose once or twice daily is preferable to giving small doses at shorter intervals.

Bronchiectasis.

Including Putrid Bronchitis.

Bronchiectasis, or dilatation of the bronchi, is not a very uncommon condition in infants and children. In most pulmonary disorders in these subjects, very slight dilatations of the bronchi may result. These have no clinical significance, and retrograde to the normal state in time. The marked dilatations are the congenital bronchiectasis and the acquired or inflammatory form.

Congenital Bronchiectasis.—This is a condition of the newly born infant which has been known to persist into adult life (Grawitz, Welch, Kessler, Fränkel). It generally affects one lung or a part of one lung. The lung structure is replaced by cystic formations which contain a serous fluid, in which are found nuclei and ciliated epithelium. The main bronchi may be cystic, with a system of minor cavities separated from the main cavity by a series of septa. In this way numerous recesses are formed. The walls of the cysts may be covered with several layers of cuboidal epithelium. No distinctive symptomatology has been reported in these cases.

Morbid Anatomy.—**Inflammatory Form.**—The inflammatory form of bronchiectasis may be sacculated, spindle-shaped, or cylindrical (vicarious). The cylindrical bronchiectasis shows the bronchus dilated into a cylindrical form. This dilatation may merge gradually or abruptly into the main bronchus. The spindle-shaped bronchiectasis is only a form of the cylindrical variety.

The sacculated bronchiectasis is the most common variety, and clinically the most important. It usually affects the smaller bronchi. A sac communicates with the trachea, and has no other outlet. The entry into the sac may be by way of a normal, a dilated, or a stenosed bronchus. If the infundibula are dilated, small cavities are formed (pulmonary vacuoles). In other cases the afferent bronchus may be obliterated, and the cystic formations are then of varying size. The wall of the bronchus leading to a cavity of this nature is in a state of catarrh, and may be thickened or infiltrated. The epithelium may be present only in spots. The infiltration may affect the walls of the alveolar septa. The mucous membrane may after a time become atrophic. The cartilages of the bronchi may also become atrophic and be replaced by connective tissue which may extend for varying distances into the lung substance, forming trabeculæ. The epithelium of the bronchi may be replaced by pavement epithelium. The mucous membrane becomes thickened or is replaced by polypoid masses. The bloodvessels finally become dilated. There may thus be formed throughout the lung small aneurismal dilatations of the bloodvessels. The remaining lung tissue may be emphysematous or sclerosed as above. The pleura may be thickened.

Etiology.—Whatever the exact cause of a bronchiectasis, there is certainly a diminished resistance of the walls of the bronchus to the inroads of inflammatory processes. In order to explain the immediate formation of these cavities, Hoffman has assumed that a stenosis of the lumen of the bronchus (as shown by Fränkel and Lichtheim), must be produced by inflammatory processes and that under these conditions the repeated attacks of coughing produce dilatation. Such stenosis may have its origin in a peribronchitis or a pneumonia causing thickening of the wall of the bronchus. Pleurisy, chronic pneumonia, croupous or catarrhal, syphilis, and foreign bodies lodged in the lumen of the bronchi may be the direct cause of a bronchiectasis. Finally, there are the forms of bronchiectasis called primary, because their etiology has not as yet been explained.

Symptomatology.—The symptoms include expectoration, a cough, dyspnœa, deformity of the chest, and fever.

Expectoration.—There is expectoration of a mucopurulent character, which cannot be differentiated from the expectoration of some forms of bronchitis. In other cases, large quantities of a fetid, purulent material are expectorated. This expectoration may at times be mingled with streaks of blood, or there may be a distinct hemorrhage. In some cases there is a fatal hæmoptysis. Sometimes the sputum is profuse, exceedingly fetid, fluid, and purulent, and will on standing separate into a serous and a purulent portion.

The **cough** may be occasional or, if the bronchiectasis exists in the apex of the lung, incessant. It is apt to be more marked in the morning, and may at that time be accompanied by the expectoration of the sputum accumulated during the night. At other times, change of position will cause paroxysms of coughing and the evacuation of large quantities of sputum.

Dyspnœa is present not only during the paroxysms of coughing, but also in the intervals, especially if there are extensive secondary changes in the lungs or pleura.

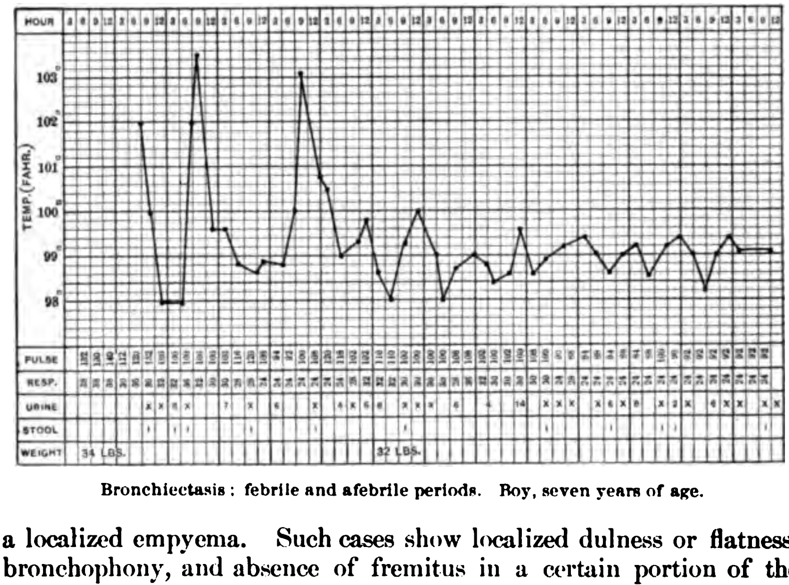
Fever of a hectic character is very likely to be present at times when the secretion in the lung accumulates. The temperature will then show a rise of a degree or more, but subsides when the lung is again cleared of bronchiectatic accumulations (Fig. 104). The rises of temperature may simulate those in empyema or tuberculosis. If abscess of the liver or kidney, endocarditis, or pneumonia occurs as a complication, the rise of temperature will be more marked.

Deformity of the chest is apt to occur in severe cases in which there is emphysema of the lung or pleuritis. In 3 of my cases there have been deformities of the fingers and toes. These, the so-called clubbed fingers, are not characteristic of bronchiectasis, since they are found in congenital cardiac disease and tuberculosis of the lung. There is pain as a result of existent pleurisy. Albuminuria may be present as a result of amyloid changes. Hæmoptyosis is generally a late symptom, but is not very common. Diarrhœa of a septic nature may occur in the course of the disease.

Complications include decomposition of the bronchiectatic accumulations, pneumonia, gangrene of the lung, emphysema, pleurisy, empyema, perforation of the lung, laryngeal disease, kidney and heart disease, liver abscess, abscess of the brain, and finally amyloid degeneration of the liver, spleen, and kidneys.

Diagnosis.—A positive diagnosis of bronchiectasis cannot always be made, especially in those cases in which there are all the signs of a localized empyema. Such cases show localized dulness or flatness, bronchophony, and absence of fremitus in a certain portion of the chest, generally at the lower portion behind. A needle, on being introduced, withdraws pus, which in the cases I have seen was mingled with air bubbles. On operation, the pleura is found to be normal. In 3 instances I found this to be true. The evidence of a bronchiectatic cavity lay in the persistence of signs and symptoms after the healing of the chest wounds. In all 3 cases the expectoration persisted in profuse quantities after operation (Fig. 105).

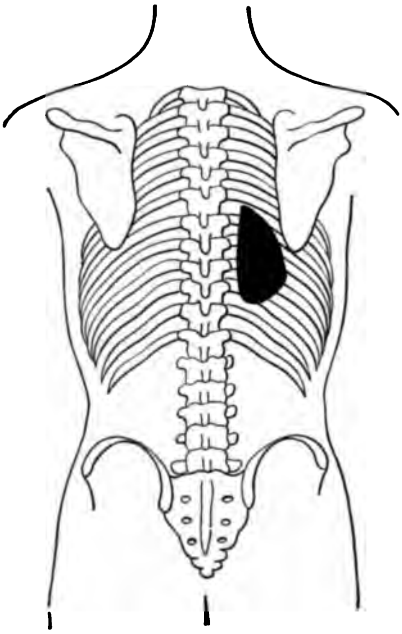
Fig. 104.



Bronchiectasis: febrile and afebrile periods. Boy, seven years of age.

The physical signs in all of my cases included a localized area of dulness or flatness, over which there was bronchophony and bronchial breathing, in some cases with gurgles. Above this area, over the base behind, there was on percussion a tympanitic note, indicating the enlarged bronchus containing air. Tuberculosis is excluded by the absence of tubercle bacilli in the sputum, but bronchiectasis and tuberculosis may coexist. In most of my cases there was a history of an antecedent attack of pneumonia. Exclusion of abscess of the lung is very difficult in severe cases in which the quantity of sputum is excessive. The bronchiectatic cavity in these cases is very large. With the bronchiectasis, there may be diffuse bronchitis and emphysema of the lung.

FIG. 105.



Showing bronchiectatic cavity in case of a girl eight years of page, with signs as noted in text.

Course.—Some of the cases in which the bronchiectasis is not marked or progressive result in spontaneous recovery. In others there may be tuberculosis, gangrene of the lung, or empyema, as complications. A fatal hæmoptysis may close the scene of this very offensive affection.

Treatment does not give very satisfactory results. It includes the inhalation of balsams of all kinds, out-of-door life in high altitudes, and surgical interference including exposure of the lung and incision of the bronchiectatic cavity. The latter is a desperate remedy; in some cases it has resulted in fatal hemorrhage and in others has not afforded relief. A cure has resulted in a few rare cases in which there was a simple cavity in the lung near the pleural surface. The injection of these cavities with drugs has also been very unsatisfactory.

V. DISEASES OF THE LUNGS.

General Considerations.—The lungs at birth are small as compared to the other organs in the chest. They grow comparatively more in the first few months of infancy; but in children they remain small as compared to the body-weight and length. Compared to the heart in volume during the first months of infancy, the lungs are as 3.5 or 4 to 1. In the later months of infancy the lungs develop more rapidly, and then the ratio of volume of the lungs to the heart is as 5.5 or 6.2 to 1.

Nose-breathing is the normal mode of breathing in infants. If an infant is studied as it sleeps, though the lips be parted and the mouth open, it will be seen that the tongue is apposed to the roof of the mouth, and that the breathing is purely nasal. If there is any obstruction in the nasal passages due to swelling of the mucous membrane, or an accumulation of nasal secretion, the infant will not only have noisy breathing, but there will be a suction backward of the tip of the tongue, and we will observe the so-called swallowing of the tongue. As soon as the nasal passages are cleared of mucus, or the swelling of the mucous membrane subsides, the infant will breathe quietly. In the young infant mouth-breathing is rarely seen. The infant has not learned as yet to overcome the obstacles to nasal breathing, should these exist, by breathing through the mouth. Later on in childhood mouth-breathing appears. Therefore adenoid vegetations in young infants may lead to greater difficulties in breathing than in older infants or children.

Movements of the Chest.—The movements of the chest may normally be irregular in rhythm; the sides move symmetrically.

In disease, especially in conditions of pressure on one side of the neck, one side of the chest may remain immobile, the other being retracted with each respiration to an exaggerated degree. I have observed this condition after operations for retropharyngeal abscess in the neck, in cases in which the nerves in this region were pressed upon or injured, thus interfering with the normal action of the diaphragm.

In effusion into one side of the chest, there is diminished motion on the diseased side. Emphysema may restrict the normal movements.

Cheyne-Stokes respiration is seen in cerebral disease. After a deep and full inspiration the respirations become increasingly shallow until they are scarcely perceptible. A deep inspiration is then taken, and the respirations become more and more marked in the ascending scale, finally reaching the original force and depth. The cycle is then repeated.

In forms of pleurisy with effusion the intercostal spaces are retracted more than is normal at each descent of the diaphragm. This may be due to adhesions. The præcordial region may be drawn inward with the recoil of the heart, as is sometimes seen in adherent pericardium.

Skoliosis of the spine may deform the chest, giving undue prominence to one side. Retraction occurs after the absorption of pleuritic effusions.

Fremitus.—The method of obtaining fremitus in children is described on page 40. It may be mentioned here that fremitus is well marked normally in the posterior axillary line and in the interscapular region.

The Normal Limits of the Lungs.—In the mammillary line on the right side to the sixth rib; in the mid-axillary line to the ninth rib. Posteriorly on the right side to the tenth rib; on the left side to the eleventh rib. Thus the limits are practically the same as in the adult subject (Symington).

The amount of lung-tissue above the clavicle cannot be mapped out in infants and children.

Resiliency of the Chest-wall.—The chest-wall in infants and children has a normal resiliency to percussion. The wall gives beneath the finger. This is a definite feature. In any disease of the chest which interposes fluid between the chest-wall and the lung this resiliency of the wall is absent. In infants and children, as in adults, there are normally:

Pulmonary resonance;
Dulness varying to flatness;
Tympanitic resonance.

Pulmonary resonance is lower in pitch than in the adult. Anteriorly over the right infraclavicular region it is less marked than on the left side; the note is also slightly higher and of shorter duration.

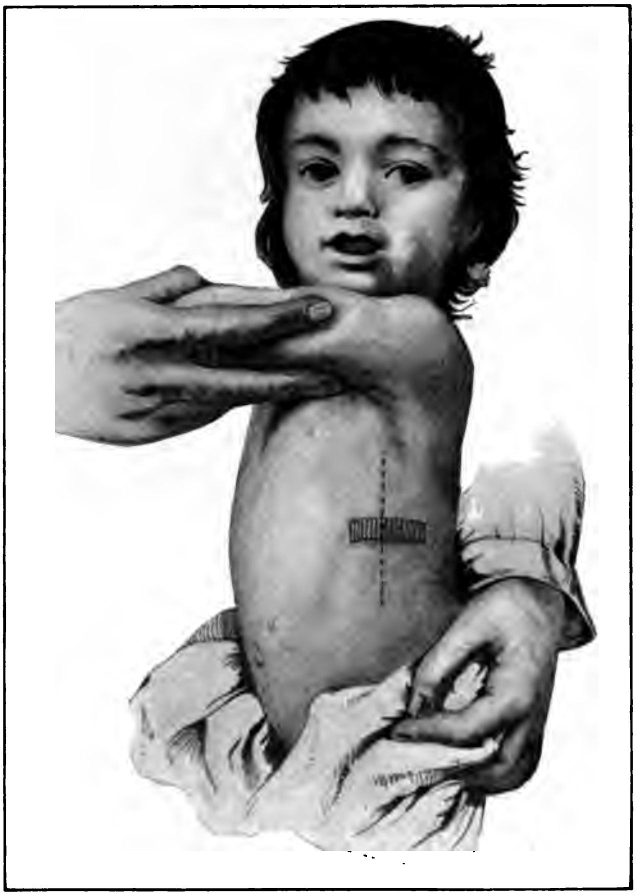
Dulness is found normally over the heart, liver, and spleen; also, *anteriorly* on the right side from the fourth to the sixth rib. From the sixth rib to the borders of the ribs the note is flat. In the mid-axillary line on the right side there is dulness from the fifth to the seventh rib; from this point to the free border, the note is quite flat. On the left side at the level of the sixth rib, just above the spleen, there is a narrow strip of relative dulness, due to the presence beneath the diaphragm of the left lobe of the liver (Fleischman) (Fig. 106).

Posteriorly the supraspinous regions give dulness, but not so markedly as in the adult. On the right side, from the level of the seventh dorsal vertebra, extending downward, there is dulness due to the liver.

Tympanitic resonance due to the stomach is found normally in the left axillary line. It may in some cases extend high up in the axilla.

Auscultation.—As a rule, there is little difficulty in obtaining the respiratory murmur and voice-sounds in infants and children—certainly not in the latter. The crying of unruly infants is useful in that it gives the fremitus and the quality of the voice-sounds. In some cases the infants are very quiet during examination, and unless they are teased into crying, definite information on these points cannot be obtained. The infant is caused to cry by gently squeezing the cheeks with the thumb and index finger.

FIG. 106.



Strip of relative dulness described by Fleischman, and found just above the spleen, supposed to be due to the presence of the left lobe of the liver. Child, two years of age.

The Breathing.—The respiratory sounds in infants and children are of an intensified vesicular quality; this so-called puerile breathing is normal and constant in children under twelve years of age. The quality of the vesicular murmur is probably caused by the better conducting qualities of the chest at this age. The elasticity of the lungs, which causes greater resistance to the inspiratory dilatation, is also a factor in producing the puerile quality of the respiratory sounds (Gutman).

Types of Puerile Breathing.—Puerile breathing in infants and children may be classified as follows:

a. The most common type is that in which the inspiration is coarse or intense in quality, while the expiration is vesicular and almost inaudible.

b. The second type of puerile breathing is that in which the inspiration and expiration are both of an intensified coarse quality.

c. The third type is that in which the inspiratory sound is low and vesicular, and the expiratory, coarse and puerile.

These types are found in infants and children at rest. If they are caused to cry, both the inspiratory and the expiratory murmur are of a coarse puerile quality. In some infants and children at rest, the inspiration and expiration are vesicular as in the adult. Puerile breathing is frequently confounded with bronchial breathing. It is, however, never tubular in quality. Bronchial or tubular breathing is marked on expiration; puerile breathing is generally so on inspiration.

During auscultation the sides of the chest are always compared. On the right side, beneath the clavicle and over the spine of the scapula, the expiratory murmur is more intense than on the left side. This should be especially remembered in cases in which disease of the right apex is suspected. The quality of the breathing in these regions approaches the bronchovesicular.

Posteriorly, the respiratory murmur may be heard as far down as the level of the eleventh dorsal vertebra. In some children the sounds are not so intense toward the base of the lung behind as higher up in the chest.

Bronchovesicular breathing is heard normally in the interscapular region in children as in adults. It has the same qualities as in the adult.

Bronchial breathing is heard normally over the trachea and upper part of the sternum. It is also called tubular, tracheal, and over the larynx, laryngeal breathing.

Forms of Dyspnœa.—Though mainly of two types, pulmonary and laryngeal, dyspnœa may be caused by pain, fever, cardiac disease, and abdominal tumors.

Pulmonary Dyspnœa.—There is not only an increase in the number of respiratory movements, but also a change in the depth of each respiratory effort. In the dyspnœa of pulmonary disease, the region at the border of the ribs adjacent to the abdominal walls (peripneumonic groove) is drawn forcibly inward at each inspiration. In emphysema with asthmatic attacks, it will be noticed that during the attack the upper part of the thorax is immobile, the inferior part being drawn inward with each inspiratory effort. The presence of fluid in one side of the chest may be suspected if the side remains immobile, or if the intercostal spaces are drawn inward with each forced inspiration. A splenic or nephritic tumor may also, by simple upward pressure, immobilize one side of the chest.

Laryngeal dyspnœa will occur in any obstructive disease of the larynx. In addition to the phenomena of the pulmonary form of dyspnœa, there is a distinct retraction of the tissues at the situation of the suprasternal notch. There may also be laryngeal or croupy breathing.

While this is true in the majority of cases, I have also seen the retraction of the suprasternal notch, described above, present in the later stages of severe forms of acute pulmonary disease, especially in children; also in cases of emphysema in the asthmatic attack.

Pain will cause an increase in the number of respiratory movements. Thus the pain of an incipient pleurisy will cause an increased number of respirations which are more shallow than is normal. Peritonitic pain will also cause the respirations to become shallower and to increase in number.

Fever will, especially in infants and children, increase the number of respiratory movements to 40 or more, without the presence of any lung disease.

Cardiac dyspnœa is seen in those diseases of the heart which cause a retardation of the pulmonic circulation. The aëration of the blood in the capillaries of the lung is considerably interfered with under these conditions. Mitral disease, stenosis, and regurgitation cause dyspnœa not only for the reason given above, but also, in the later stages, on account of the bronchitis which is the result of the cardiac disease. Anæmia of cardiac disease is also accompanied by a slight dyspnœa, which is especially marked in children. The slightest exertion will sometimes cause angina and dyspnœa in children suffering from a slight cardiac lesion.

Ascites and **abdominal tumors**, or enlarged organs, such as the liver or spleen, will cause dyspnœa, especially when patients are in the recumbent position.

In weak infants a few days old, who are the subjects of atelectasis and pneumonia, the upper part of the chest-wall moves very little, while the inferior portion of the chest and the upper part of the abdomen (peripneumonic groove) are drawn inward at each inspiration.

Pneumonia.

Lobar Pneumonia.

(*Fibrinous Pneumonia, Croupous Pneumonia or Pneumonic Fever*).

Lobar pneumonia or fibrinous pneumonia is an acute infectious disease, caused in the majority of cases by the Diplococcus pneumoniæ (Fränkel). A few cases are caused by the Bacillus pneumoniæ (Friedländer); others, by the Streptococcus or Staphylococcus pyogenes.

Occurrence.—Lobar pneumonia occurs as a primary disease or may complicate typhus fever, typhoid fever, influenza, rheumatism, malarial fever, erysipelas, osteomyelitis, meningitis, and nephritis. According to Keller, from 58 to 62 per cent. of all lobar pneumonias occur among children, the frequency among boys being greater (55.9 per cent.). Fully two-thirds of the cases occur during the winter and early spring. Pneumonia of any variety, and especially of this form, may occur in groups of persons or in small local epidemics. Without doubt, certain houses and rooms harbor the pneumonia poison for some time, as is evinced by the repeated occurrence of cases in certain places (Jürgensen). Cold favors the development of pneumonia by reducing the resistance of the economy to the invasion of bacteria, but it cannot be regarded as a cause of the disease.

Age.—Lobar pneumonia may occur at any age of infancy or childhood. Von Jaksch has shown that it occurs among young infants. My own experience confirms this statement. Out of 839 of my cases of pneumonia of all types, 582, or 69 per cent., occurred before the end of the second year; the greatest frequency was between the first and second years (282 cases). From birth to the sixth month the frequency is less than from the sixth month to the end of the second year.

Sex.—The male sex shows the greater number of cases (436 males, 403 females). Of 147 cases of carefully observed lobar pneumonia, 89 were males and 58 females.

Seat of the Disease.—Jürgensen shows that in 162 cases, both lungs were affected in 7.4 per cent. The right lung only was affected in 43.2 per cent. of the cases. When the right lung was attacked, the lower lobe was generally the seat of the disease (25.3 per cent.). The lower lobe of the left lung was consolidated in 35 per cent. of the cases.

Of 217 of my cases of lobar pneumonia, the right lung was involved in 124 cases and the left in 93; the upper right lobe was involved in 74 cases; the upper left, in 35. The upper lobe of either lung was involved in 109 cases, as against 100 cases of the lower lobes. The middle right lobe was involved in only 8 cases.

	Upper lobe.	Middle lobe.	Lower lobe.
Right lung	74	8	42
Left "	35	. .	58

Pneumonia of the upper lobe is more frequent in children than in adults. According to Jürgensen, the greater frequency of pneumonia in the right lung may be attributed to the larger size of the right bronchus and the more direct communication with the lung.

Morbid Anatomy.—Lobar pneumonia in infancy and childhood is, as in adult life, distinguished by the occurrence of a fibrinous exudate in the alveoli of the lungs, bronchioles, and lymph-spaces. This exudate is composed of desquamated epithelium, leucocytes, red-blood cells, and fibrin. The proportion of leucocytes, red blood-cells, and fibrin varies greatly at different stages of the affection. A fluid exudate may be present if the quantity of fibrin is small. In such cases there is a lobar catarrhal process or an inflammatory œdema of the lung. The exudate begins with congestive hyperæmia. The lung is dark red and of increased consistency. With the appearance of coagulation there is produced a condition of hepatization in which the lung is solid, and has the appearance of liver. The bloodvessels are filled with red cells. If the vessels are less engorged, the lung has a grayish tint. This later stage, called gray hepatization, is the condition most frequently seen at autopsy. The hepatized lung does not contain any air, and on section shows a granular surface, the granules being the so-called pneumonic granules of the later stage of the disease. The pleura is as a rule inflamed. It is without lustre and may be thickened and covered with fibrin. There may be considerable serous or seropurulent exudate in the pleural cavity. The extent of hepatization varies. It may involve a whole lobe, part of the lobe of a lung, or parts of both lungs. On inspection of the surface of a section, small yellow areas may be seen in the hepatized portions. These are areas poor in fibrin, and correspond to the situation of the bronchioles of the lung.

The bronchial nodes may be red and swollen, the bronchi being the seat of inflammation. The bronchioles may be filled with fibrin and red blood-cells.

Resolution occurs on from the seventh to the tenth day of the disease. At this time liquefaction of the inflammatory products which are eliminated by expectoration occurs. Complete restoration of the lung to the normal may occur between the second and the fourth week, at which time the periphery of the alveoli may be found to be rich in cells. There may still exist catarrhal processes which have succeeded the fibrinous changes. The pleura may remain thickened and be the seat of adhesions.

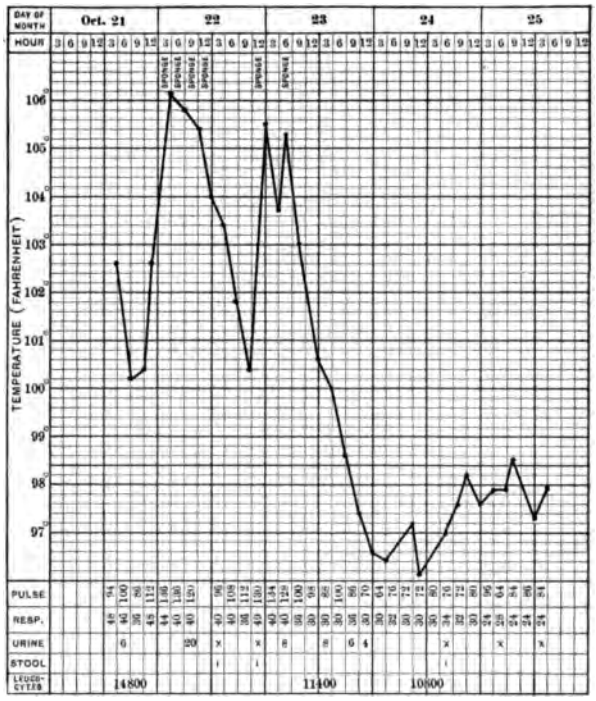
An unfavorable or malignant ending, such as gangrene or suppuration, is rare, and is as a rule due to some mixed infection favored by an old bronchiectasis or putrid bronchitis. Unless a tuberculous infection occurs, caseation in lobar pneumonia is unknown. Induration of the lung, cirrhosis or carnification, is a peculiar condition which may occur from the fourth to the tenth week. The lung assumes a beefy red appearance and is tough, hyperæmic, and infiltrated with small round cells. The alveoli enclose a large number of connective-tissue cells. There is a proliferation of newly formed bloodvessels in the septa of the lung. The bronchial, peribronchial, and pleural tissues are proliferated. Induration of the lung by pleural adhesions results. The alveoli of the lung may be replaced by connective tissue and epithelium. Induration may take the form of bands of connective tissue, which may extend from the pleura into the lung, enclosing areas of lung-tissue.

Bacteriology and Etiology.—The pneumococcus of Fränkel is now recognized as the etiological factor in lobar pneumonia. As has been mentioned, the Bacillus pneumoniæ of Friedländer is found in a small number of cases, with the pneumococcus or with other bacteria. The Streptococci pyogenes and the Staphylococcus pyogenes are sometimes found, as well as the Bacillus typhosus. In the cases of secondary infection, the Diplococcus pneumoniæ or the Staphylococcus pyogenes is found. In the majority of fatal cases, Kohn found the pneumococcus circulating in the blood. The cases which show the diplococcus in the blood and which recover, do so with complications. In a recurrent pneumonia of infancy, Perutz found an osteomyelitis of the joint, caused by pneumococci. In one of my cases which was followed by bilateral empyema, there was a peri-articular abscess containing pneumococci. According to Landouzy and Netter, the pneumococcus is capable of producing suppuration without the intervention of streptococci or staphylococci. Cases of severe icterus are due to the hæmolytic action of the pneumococci on the blood. Gaillard has shown that the enteritis in pneumonia is caused by pneumococci.

Symptomatology.—There are forms of fibrinous or lobar pneumonia which present the same symptomatology in children as in the adult. On the other hand, certain sets of symptoms referable to the nervous system and intestinal tract, as well as the character of the variations in temperature, are peculiar to infancy and childhood.

The disease may be ushered in by a chill, which may be severe or only amount to a sensation of chilliness. Susceptible subjects may, with the rise of temperature, be attacked with convulsions. Other patients pass into a stage of delirium lasting for days. Cases of pneumonia ushered in with cerebral symptoms are apt to mislead the physician, especially if meningitis has been recently prevalent. There are also cases, especially in children, in which there has been a preceding bronchitis. These should not be regarded as being of necessity cases of bronchopneumonia. Sometimes the chill is coincident with a sharp attack of enteritis. The character of the invasion will thus vary with the severity of the infection and the susceptibility of the subject. After the initial chill, there is in the simple cases a sharp rise of temperature. The height of the fever varies, and in young infants is apt to mount to 106° F. (41.1° C.). There are cough and considerable dyspnœa, varying with the extent of lung involvement. In infants and children the dyspnœa is quite apparent to the eye of the observer, and will prompt him to surmise that the lung may be involved. Older children have a distressed expression. In cases in which sopor is present, the dyspnœa is apt to be more evident than in those cases in which this cerebral symptom is absent. This apparent dyspnœa is only relative. A conscious patient does not show this dyspnœa as much as one who is unconscious.

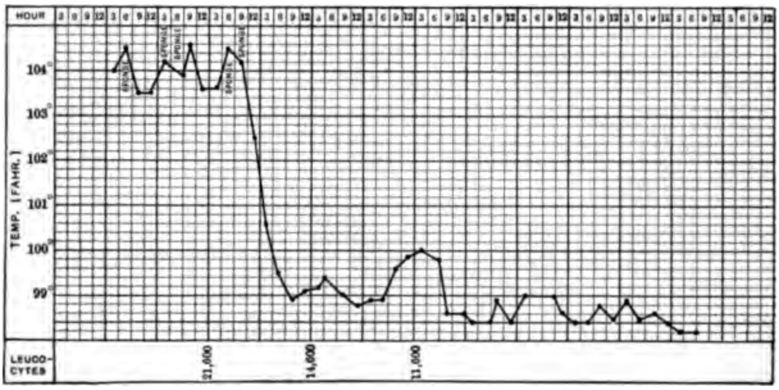
FIG. 107.



Lobar pneumonia; pseudocrisis and crisis. Leucocyte count before and after crisis indicated. Boy four years of age.

The patient complains of pain, which is in many cases referred to the side affected. In younger children the pain is quite frequently referred to the epigastrium, but sometimes to the region of the abdomen low down, or to the right side of the abdomen low down over the situation of the vermiform appendix. Pain is apt to be referred to this region in cases of lobar consolidation of the lower portion of the right lung. These are often, in the early stages, diagnosed as cases of appendicitis. The face is pale or quite flushed. The dyspœna may be slight, but is quite marked in some severe cases. Even if both lungs are involved, it may not be intense. There is a cough. In older children there is expectoration of rusty sputa. Infants and young children swallow the sputum. Infants cry with each paroxysm of coughing; older children complain of pain. Sometimes infants and children vomit with each attack of coughing. After the fever has persisted with these symptoms for from five to nine days, there occurs in the vast majority of cases a fall of the temperature—the so-called crisis—which may take place within from three to six hours, or may extend over thirty-six hours. The fall of temperature may be followed by a temporary rise of a few degrees (Fig. 107)—the so-called pseudocrisis; within a few hours it then falls to the subnormal, where it remains for a few days after the crisis, finally rising to the normal and remaining at that point throughout convalescence. The temperature may fall by lysis, that is to say, by reaching with gradual remissions the normal, or as a rule the subnormal, within from forty-eight to seventy-two hours.

FIG. 108.

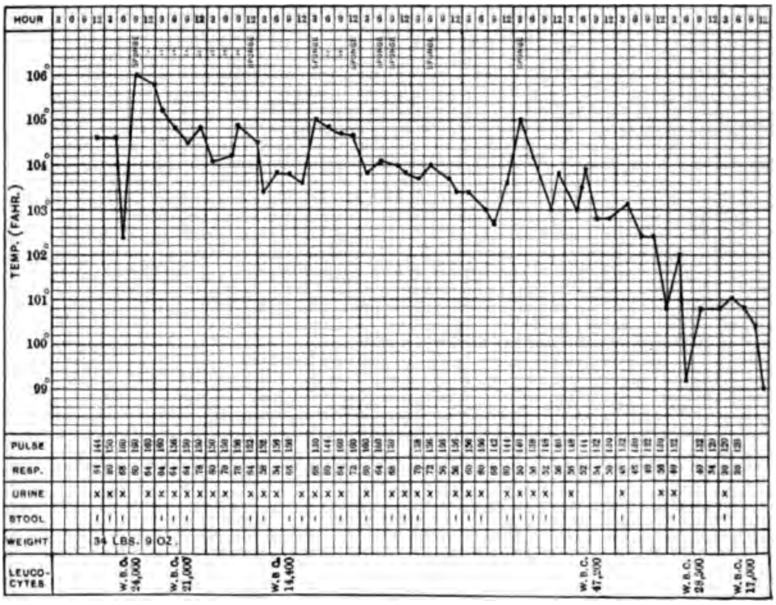


Lobar pneumonia, right lung, lower lobe. Crisis on the eighth day. Leucocyte count indicated. Female child, two years and five months of age.

Consideration of Individual Symptoms.—THE TEMPERATURE.—The temperature-curve in lobar or fibrinous pneumonia may be of several distinct types. In the majority of cases the temperature remains persistently high for the whole period of the illness. There are morning remissions of a degree or more, but the afternoon or evening rise may reach 104°, 105°, 106° F. (40°, 40.5°, 41.1° C.). In a typical case the morning remissions are not so great as those in pneumonia of the bronchopneumonic type. The crisis is not as a rule preceded by a rise. The drop of the temperature at the crisis in a fairly typical case may begin at 9 A.M., and the temperature may be subnormal at 9 P.M. of the same day (Fig. 108). In another form, crisis may be rapidly followed by a temporary rise in the temperature, not due to any reinfection of the lung, but to a slight post-pneumonic toxæmia. The temperature will in such cases reach the subnormal within thirty-six hours.

Another very distinct form of temperature-curve is the remittent. This temperature-curve is at first glance exactly similar to that of bronchopneumonia. The remissions in the morning may reach the normal within a fraction of a degree. Such cases may also show at the terminal end of the curve a critical drop to the normal. In other cases the fall of temperature at the beginning of convalescence takes place by what is known as lysis (Fig. 109). In other words, the temperature reaches the normal or subnormal by remissions of temperature in a gradually descending scale extending over two or more days. Some cases show a remission of the temperature which begins at the ninth day of the disease, and is not completed until the fifteenth day. This is occasionally seen in cases in which there are apparently no complications. The more common type is that in which the lysis begins on the seventh or eighth day, and is completed in two or three days. Of 57 cases of lobar pneumonia in which a reliable history could be obtained, the temperature fell by crisis in 36

FIG. 109.

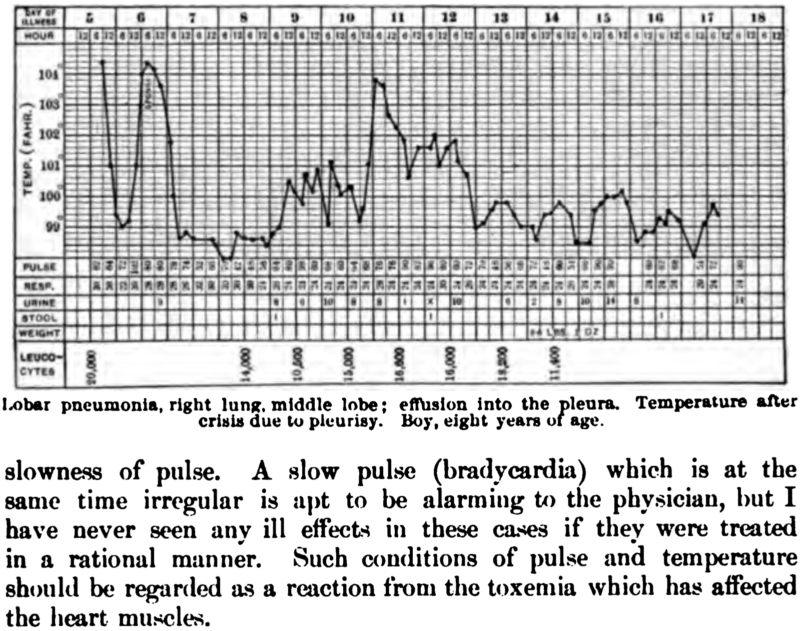


Lobar pneumonia, right lung, lower lobe; temperature falls by lysis. Leucocytosis indicated in the chart. Female child, four years of age.

and by lysis in 21 cases. The crisis, as a rule, occurs from the fifth to the ninth day of the disease (60 per cent. of my cases). After the lysis or crisis there may be a slight daily rise in temperature of a degree or even less, probably indicative of a very mild form of post-pneumonic pleurisy. The temperature in such cases falls gradually, and in four or five days reaches the normal (Fig. 110).

The subnormal temperature after the crisis or lysis is quite a common phenomenon. I have learned not to fear this symptom, but to regard it as favorable (Fig. 111). A subnormal temperature may persist for days, or even a week or longer, and not uncommonly, especially in fibrinous pneumonia which has run a sharp or moderately severe course, is accompanied by irregularity or abnormal slowness of pulse. A slow pulse (bradycardia) which is at the same time irregular is apt to be alarming to the physician, but I have never seen any ill effects in these cases if they were treated in a rational manner. Such conditions of pulse and temperature should be regarded as a reaction from the toxemia which has affected the heart muscles.

FIG. 110.



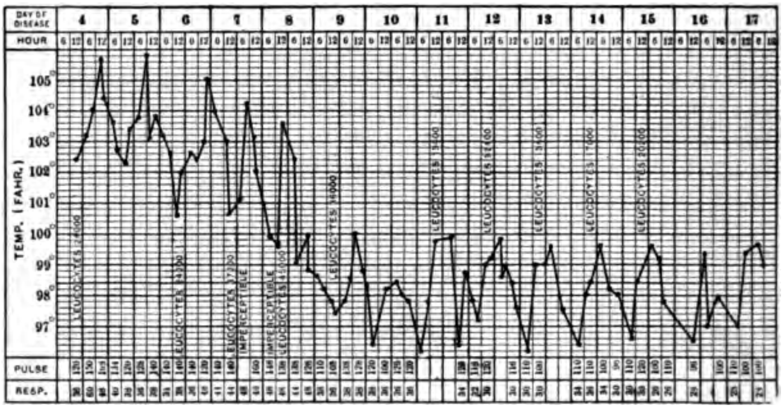
Lobar pneumonia, right lung, middle lobe; effusion into the pleura. Temperature after crisis due to pleurisy. Boy, eight years of age.

Chills, or chilly sensations followed by a rise of temperature during the course of the disease, are in most cases accompanied by physical signs of an invasion of a new area of lung. This should at least be kept in mind, especially if the rise of temperature is abrupt.

At the crisis in lobar pneumonia I have, in exceptional cases, seen the temperature drop within an hour from 103° to 94° F. (34.4° to 39.9° C.) and the pulse to 48; within an hour the temperature rose to 96° F. (35.5° C.) and the pulse to 70. The temperature gradually rose, so that within seven hours it was again 99° F. (37.2° C.) in the rectum, the pulse 96. The symptoms of mild collapse may accompany the pronounced fall.

THE COUGH.—Some infants and children cough very little; in others the cough is a very harassing symptom. There is no sputum even with the older children, or only after the crisis; pain accompanies the cough, and may be suspected if the infant or child cries when it coughs. The pain is referred to the side of the chest, to the epigastrium, or to the region of the appendix. The pain referred to the appendix in cases of lobar pneumonia is probably radiated from a diaphragmatic pleurisy.

FIG. 111.



Lobar pneumonia, right lung, upper lobe: remittent temperature-curve; prolonged subnormal temperature intermittent in character; recovery. Female child, two years and six months of age.

DYSPNŒA.—Infants and young children show marked dyspnœa. The alæ nasi are dilated and the peripneumonic groove is depressed with each inspiration. In very severe dyspnœa in young infants, there may be a drawing inward at the suprasternal notch. This occurs even in the absence of any laryngeal disturbance, and frequently simulates laryngeal stenosis.

NERVOUS SYMPTOMS.—The cerebral symptoms may at the outset simulate those of cerebrospinal meningitis. There are delirium, rigidity of the muscles of the neck, and even opisthotonos. There may be no true meningitis. Older children may have a low, muttering delirium during the whole course of the disease. Near the crisis and just before the fall of temperature, I have in a few cases seen maniacal delirium, in which the patients were very noisy and attempted to get out of bed. I have seen cases of melancholia with crying spells during convalescence in female children, and also in boys. These symptoms all subsided in time and the patients were eventually fully restored.

THE BLOOD.—It has been noted by Tumas and von Jaksch that in pneumonia of the fibrinous variety there are a marked leucocytosis and an increase in the multinuclear leucocytes, which is especially marked at or near the crisis. The proportion of leucocytes to the red blood-cells in the cubic millimetre may reach 1 : 40 to 1 : 70. Ehrlich believes this leucocytosis to be a very constant occurrence in typical pneumonia. Billings has investigated the relationship of the leucocytosis to the prognosis more fully. His work will be referred to in the consideration of the prognosis. My own experience covers about ninety cases of fibrinous and bronchopneumonia, examined with reference to leucocytosis. Leucocytosis is present in both forms of pneumonia in infancy and childhood, but is more marked in the fibrinous forms, the number of leucocytes to the cubic millimetre being about twice as great as in the catarrhal forms. There was marked leucocytosis in the fatal cases of both forms of pneumonia. The increase of the leucocytes in the fibrinous forms was especially marked at the time of the crisis. In the bronchopneumonic forms the leucocytes were also high at or about the time of the drop in temperature. The diminution of the number of leucocytes was in both forms marked either just previous to or after the fall in the temperature. From the observations of Billings and Ewing, it must be concluded that leucocytosis is a favorable sign in fibrinous pneumonia. It does not, however, as Ewing believed, bear any exact ratio to the extent of lung involved. I have found a much higher percentage of leucocytes to the cubic millimetre in children than Ewing found in the adult. This is probably due to the fact that any leucocytosis is more marked in infants and children than in the adult subject. The absence of leucocytosis is certainly a grave prognostic sign, but the presence of marked leucocytosis in children does not in my experience preclude a fatal issue.

Physical Signs.—The signs obtained by physical examination of the chest in fibrinous pneumonia of infants and children resemble those of the same condition in the adult. In forms of bronchopneumonia or catarrhal pneumonia in which areas of considerable extent are consolidated the signs will closely resemble those obtained in the fibrinous form. The physical signs of lobar or fibrinous pneumonia are classified as those of the first, second, and third stages of the disease.

First Stage, Stage of Engorgement of the Lung.—On INSPECTION the signs of dyspnœa above noted are found.

PALPATION at this stage will in an uncomplicated case give no signs, even over the affected area. If bronchitis complicates the case, rhonchal fremitus may be obtained. At this stage the difference in fremitus between the affected and the unaffected side of the chest is not perceptible.

AUSCULTATION.—In the first stage of the disease auscultation may discover a rude respiratory murmur on the healthy and diseased sides which is more marked in the latter and on inspiration. The pathognomonic sign at this stage is the crepitant râle, which is sometimes easily found and is at others very elusive. It may be present before an attack of coughing, and disappear after the bronchi have been cleared, and is, as a rule, heard over a very limited area. It is therefore necessary to examine the chest very carefully in front, behind, and in the axillary line for this sign, before deciding positively as to its presence or absence. It may be present for a few hours only.

PERCUSSION will at this period give slight dulness over the affected area of lung. The dulness may be slightly tympanitic. This is caused by the fact that at the outset of consolidation there is still some air in the affected area. Under these conditions there may be what is known as tympanitic dulness. This condition is especially found in young infants, in whom the chest-wall is thin, and in whom sounds are very well obtained by gentle percussion.

The Second Stage, Stage of Consolidation.—If the lower portion of the right lung is affected, we shall get by palpation in front over the upper part of the chest nothing abnormal; over the lower part of the chest in front there will be an increase of the vocal fremitus, which is also apparent behind. Percussion over the upper part of the right lung will give a vesiculotympanitic note in front and behind. The unaffected side will give normal pulmonary resonance. In exceptional cases the percussion-note over the upper lobe of the lung in front may give the so-called cracked-pot sound. In front, behind, and in the axillary line over the lower lobe which is affected there is dulness—not at first complete. When consolidation is complete, the dulness is quite marked. In cases in which some pleuritic effusion exists over the consolidated area behind, the percussion-note may be quite flat. In cases in which the upper lobe is consolidated there will be signs of consolidation, while lower down the note is exaggerated or vesiculotympanitic over the unaffected mid-region of the lung, and over the base there will also be marked dulness. This lower area of dulness should not be regarded as a sign of consolidation. It is really due to the accumulation of a small amount of serous effusion in the lower part of the pleural cavity as a result of the complicating pleurisy.

AUSCULTATION will in this stage give bronchial voice and breathing over the affected area of the lung; over the unaffected lung the respiratory murmur, especially the inspiratory sound, is harsh. This harsh inspiratory sound is quite common in children, and is frequently mistaken for bronchial breathing. Bronchial breathing is tubular in quality on inspiration and expiration. In this stage, if the upper lobe of the lung is also involved and there is some pleuritic effusion in the chest, the respiratory murmur may be much weakened over the lower region of the chest behind.

The voice also has a tubular or bronchial quality over the consolidated area. The intensity of the voice may be diminished over the lower portion of the chest if pleuritic effusion is present with consolidation of the upper lobe. Pleuritic râles may in this stage be heard over the whole side of the chest.

Third Stage.—The third stage, that of resolution, is sometimes delayed, some days elapsing after the crisis before appearance of the sign pathognomonic of this stage—the so-called râle redux. This râle has the same qualities as that heard in adults at the same stage. In children it is sometimes present for only a short time, and is not heard over any considerable area of the lung. I have known the temperature to be subnormal for two days or more before its appearance. The other sign, which is less important, is a distinct diminution of the fremitus until it reaches the normal intensity over the affected area of lung. The percussion-note becomes less dull, assuming the vesiculotympanitic quality. Repeated auscultation reveals, in addition to the râle redux, a gradual return of the voice and breathing to the normal, which sometimes takes weeks. The tubular quality of the voice and breathing over the affected area of lung may persist long into convalescence. It is probably not caused by any actual persistence of consolidation, but by a continued hyperæmia of the lung. The lung under these conditions is denser and conducts sounds from the bronchi with greater intensity than the healthy lung. If pleurisy has been present to any extent, there may, after the disappearance of the signs of consolidation, be signs of dry pleurisy or those of effusion.

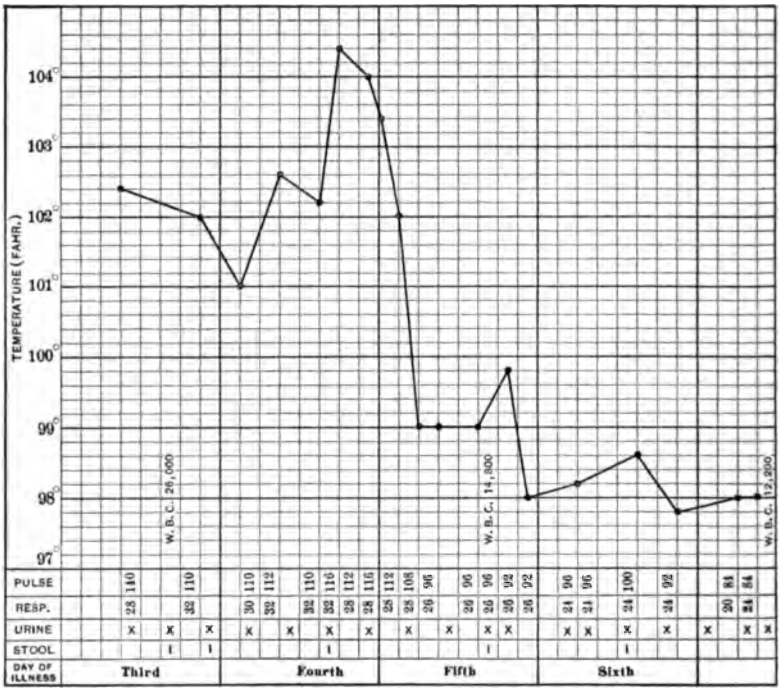
Pneumonia of an Unusually Short Course.—Leube and Weil have recorded in the adult typical pneumonia of the fibrinous variety and of very short duration. Some of these cases exhibit the chill, fever, pain, and crisis, with other signs of physical involvement of the lung, within twenty-four to thirty-six hours. Jürgensen has recorded short lethal pneumonias of the fibrinous variety in the adult. The cases of Levy and Jürgensen were fatal within twenty-four to thirty-six hours. I have never met such cases of fibrinous pneumonia in children, but have seen lobar pneumonia with a history of short duration (Fig. 112). In cases running a very short course there is doubt as to whether the signs obtained over the chest may not have been connected with a preceding attack. Henoch has, however, met a few cases which ran a rapidly fatal course, with the whole symptomatology of lobar pneumonia including physical signs, in forty-eight hours.

Complications.—Among the complications of fibrinous pneumonia in infants and children are otitis, pleurisy, pericarditis, endocarditis, empyema, and meningitis arthritis and osteomyelitis. Some writers record peritonitis; I have not met a case. Gastro-enteritis is quite a common complication.

Otitis is common, its frequency varies in different epidemics.

It affects younger children and infants more frequently than older subjects. The temperature in these cases becomes more markedly remittent and remains higher for a greater length of time than in the uncomplicated cases. I have frequently suspected otitis from a study of the temperature-curve, which is not, however, an altogether reliable guide. Suppuration in the pleura will give a similar curve. Therefore, in a concrete case of persistent high temperature-curve with morning remissions, otitis should be suspected, but not positively diagnosed without careful exclusion of other complications.

FIG. 112.



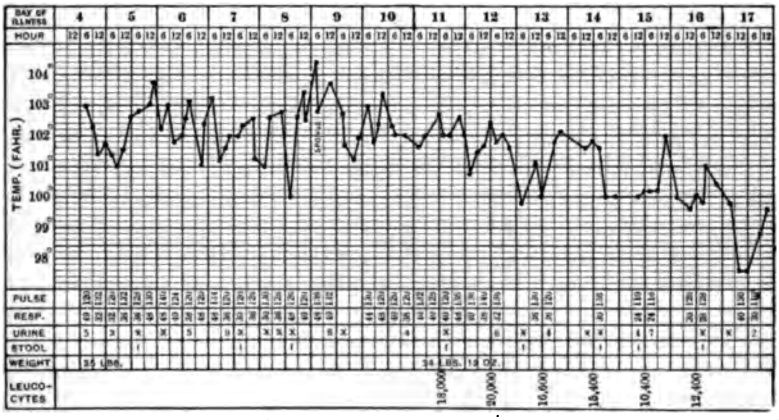
Lobar pneumonia, midregion of the right lung; crisis on the fourth day of disease. Boy, seven years of age. (Author's case.)

Otitis as such does not seem to give any striking symptoms of pain. The patient may without warning present perforation of the drum of one or both ears and a purulent discharge. The temperature will then fall to the normal. Diplococcus pneumoniæ has been found by a number of observers in this discharge. The otitis is of a benign nature.

Meningitis occurs in a number of cases, and may usher in the disease. I have seen it persist for weeks. The prognosis in this form of meningitis, if it assumes the cerebrospinal type, is graver than when it occurs as a primary disease, with the intracellular diplococcus of Weichselbaum as a causative factor. Netter seems to have met a larger number of cases of the pneumococcus form of meningitis than any other author. The cases of meningitis complicating pneumonia should not be confused with those presenting cerebrospinal symptoms. The cerebrospinal symptoms seen at the outset or at the crisis in some cases of pneumonia do not last for any great length of time, and do not present the true symptoms of meningitis.

Pleurisy and Empyema.—Many cases of fibrinous pneumonia show a dry pleurisy sometimes persisting for a long time after convalescence. Of greater moment are the cases of pleurisy with effusion, which follow a lobar pneumonia. In these, there is always the danger that the exudate may eventuate in an empyema. The duration of the exudate is no guide in determining whether it is of a serous or a purulent nature. It is frequently found that after a pneumonia has run its course the temperature remains raised a degree or more toward evening. Such a rise in temperature may, in the absence of signs of fluid, indicate a dry plastic pleurisy (Fig. 113). On the other hand, if there are signs of fluid and the temperature-curve shows irregularities of rise, empyema may be present. I have met empyema without any rise of temperature in infants who showed the physical signs of fluid in the chest. These points will be more fully discussed under the head of Empyema.

Fig. 113.



Lobar pneumonia, lower lobe, left lung; complicating pleurisy; temperature falling gradually to the normal. Leucocyte count indicated. Boy, five years of age.

Pericarditis.—I have seen pericarditis in infants who died of a fibrinous pneumonia, but the diagnosis was not made during life. Von Jaksch notes such cases. In older children, pericarditis is a complication found in cases of fibrinous pneumonia which have simultaneously developed empyema. Such cases are very uncommon. In the form of pericarditis which I have seen in infants, the quantity of effusion has not been sufficiently great to enable a diagnosis to be made with certainty, and the râles in the lung obscured the friction-sounds in the pericardium if they were present. Purulent pericarditis in these subjects is very fatal under such conditions. In older children I have seen pneumonia combined with a fibrinous pericarditis pure and simple, without fatal issue.

The **prognosis** of lobar pneumonia varies within certain limits. Text-books give statistics taken from hospital cases, notably the most unfavorable material. Henoch gives the mortality of his cases at 5 per cent.; Baginsky, at 8 per cent.; Holt, at 12 per cent.; my own hospital cases during the past year showed a mortality of 8 per cent. On the other hand, in private practice death from an acute fibrinous pneumonia rarely occurs in a child previously healthy and living in good surroundings. The mortality is influenced by the season of the year, being greater from December to February, and by the presence of an epidemic. If pneumonia is prevalent during an epidemic of influenza, the mortality will increase. Pericarditis or complicating empyema influence the death-rate. The previous condition of the patient, the mode of feeding (whether by the breast or the bottle), and a rachitic or marantic condition, affect the prognosis. The age of the patient is also an important factor. Infants under one year of age are in greater danger than older ones. The prognosis is best from the third to the tenth year. The younger the bottle-fed baby, the more serious the complication of empyema. In making a prognosis in any concrete case, the physician should be guided by the extent of lung involvement and the general condition of the circulation. If one lobe alone is involved and there is an absence of bronchitis in the unaffected lung, the outlook is good. If the heart action is good and there is an absence of cyanosis, recovery can be predicted even if the temperature be high. If, on the other hand, the lysis or crisis is delayed and the dulness or flatness involves a whole side of the chest, in the presence of signs of a weak heart the prognosis should be made with caution. Meningitis of pronounced type is grave. Pericarditis in young infants and children is a complication invariably fatal. Where facilities exist, a leucocyte-count should be taken every second day, accompanied by a differential count of leucocytes. A very low leucocyte-count, with marked signs of pneumonia and a high temperature, is a grave prognostic sign, though such cases may recover. On the other hand, a continued high leucocyte-count, as has been pointed out, may be present with extensive inflammation of both lungs, and death may ensue.

The **diagnosis** of lobar pneumonia in infancy and childhood ordinarily presents few difficulties, but is not easily made if in addition to the pneumonia there is an effusion in the chest. The diagnosis should never be made early in the disease without positive signs.

The crepitant râles sometimes escape observation. The physician should then wait for the appearance of dulness or bronchial voice and breathing before arriving at a conclusion as to the presence or absence of consolidation. Cases of influenza with a harassing cough are frequently diagnosed as central pneumonia. A pneumonia which is central will give physical signs. If after the time set for the crisis or lysis, the temperature persists and becomes remittent, careful examination should be made for evidences of fluid in the chest. The nature of the fluid should be determined by exploration with the aspirating needle, if the fever does not subside and if the dyspnœa increases. A chest effusion in infants and children is apt to be purulent.

The cerebral cases present difficulties of diagnosis. Convulsions, delirium and rigidity of the neck, accompanied by high fever and a cough, with increase of the pulse-rate and the number of respirations, indicate the necessity of making a very careful examination of the chest.

In cases which begin with a lobar pneumonia, typhoid fever may be suspected if, after the first days of illness, a roseola or an enlargement of the spleen develops with a continuance or gradual rise of temperature. In such cases the presence of an epidemic of typhoid fever and the Widal blood reaction will be of service in clearing up the diagnosis.

The **treatment** of lobar pneumonia is pre-eminently expectant. The disease is self limited, and complications cannot be prevented. The temperature should be treated within certain limits, and the heart and the strength of the patient supported. The temperature should be treated not with a view to its actual reduction, but in order to mitigate its ill effects. Infants and children will be less affected by a temperature of 103° F. (39.4° C.) lasting during the time while a pneumonia runs its course than by the same temperature in typhoid fever. The toxæmia of pneumonia is of a more benign character. Cold applications are relied on to reduce the temperature.

Hydrotherapy.—Sponging is efficient in cases in which the temperature does not generally range above 104° or 104.5° F. (40° C.). The younger the infant the less energetic need it be, for a temperature of 104.5° F. (40° C.) is not high for an infant under two years of age. I content myself with sponging of the body with water at 80° F. (26.6° C.), to which some alcohol has been added. If the temperature remits a degree or more during the twenty-four hours, there will be less need of sponging. The temperature should never be taken more often than every three hours. If it is above 103.5° F. (39.7° C.), the patient is sponged for fifteen minutes and then given absolute rest for three hours. Frequent sponging is pernicious. Some infants when sponged with water at 80° F. (26.6° C.) become blue, the pulse becoming rapid and thready. With these subjects a warm bath at a temperature of 105° to 107° F. (40.5° to 41.6° C.) is stimulating. It supports the strength and certainly lessens the ill effects of the temperature, although it may not reduce it palpably. I do not use the full cold bath in the treatment of lobar pneumonia in infants and children. If the temperature reaches 105°–106° F. (40.5°–41.1° C.), a full bath of the temperature of 85°–90° F. (29.4°–32.2° C.) or higher may be given, certainly never lower.

One of the most useful methods of hydrotherapy in the treatment of pneumonia in young infants is the so-called chest compress (page 61). These compresses renewed every hour will cause the restlessness to diminish, the heart action to improve, and the patient to fall into a quiet slumber. The actual reduction of temperature is not so marked as the favorable effect on the general condition of the patient. The application of compresses is discontinued if the temperature falls below 103° F. (39.4° C.).

Medicinal Treatment.—The heart action if good needs no attention. At most, a limited amount of wine or whiskey is administered. Infants may receive half a drachm (2.0) every few hours; older children, a drachm (4.0). Whiskey should not be given as a routine remedy. If the temperature is high necessitating hydrotherapy, and the pulse is above 120, whiskey should be given. If the pulse is high, 150–160, a few minims of the tincture of digitalis may be given to older children. Younger children rarely need more than half a minim every two to three hours. If the pulse-rate is reduced after the administration of digitalis, the drug should be discontinued before the pulse drops below 100. There is no doubt that its effect is more cumulative in some subjects than in others.

Strychnine is of value in the treatment of pneumonia, not so much in the cases with rapid as in those with slow and irregular pulse. Infants will bear grain $\frac{1}{200}$ to $\frac{1}{150}$ (0.0003 to 0.0004) every three hours, for days.

Caffeine is of great value in the treatment of irregularities of the heart which indicate a myocarditic process. The pain is the result of a pleuritic process.

The local application of iodine or mustard paper is an efficient counter-irritant. If the cough is troublesome, codeine in moderate dosage is the most useful remedy.

I never make use of morphine with infants and children. In young infants the milder preparations of opium, such as camphorated tincture or the wine, are most useful. Four minims (0.25) of the camphorated tincture of opium every two or three hours will be found efficient in children under two years of age. To older children a small dose of codeia may be given several times daily if needed. The aim is to alleviate the pain and cough.

The bowels should be evacuated daily; for this purpose hydrarg. cum creta is one of the best remedies. Grain v (0.3) may be given. Infants should receive an enema daily. If gastro-enteric disturbances ars present, the giving of milk should be discontinued and the same procedure followed as in primary gastro-enteritis.

Tympanites is sometimes troublesome, especially in young children. The best remedy is a high enema twice daily of salt solution, to which one or two teaspoonfuls of peppermint-water have been added. The passage of a soft catheter is not effective, nor are the turpentine stupes of any value.

The delirium, sometimes amounting to an acute mania, which appears just before the crisis in some cases, is best controlled by rectal administration of bromide of potassium and chloral hydrate. I have sometimes been forced to keep the patient under the influence of these drugs for a few days. The post-pneumonic melancholia seen in children is best treated by the administration of strychnine and the enforcement of perfect quiet.

Should signs of extreme cardiac weakness set in with threatening œdema of the lung and paralysis of the right ventricle, nitroglycerin is of great value. Infants will bear grain $\frac{1}{200}$ (0.0003) every three hours. If in these cases cyanosis is present, oxygen is administered, preferably that containing 20 per cent. of nitrous oxide. It is given to infants, every half hour for five or ten minutes at a time by means of a cone.

Hygiene.—The patient should be isolated if possible. The room should be ventilated and its temperature kept at 68°–72° F. (20°–22.2° C.).

The sputum should be received in pieces of gauze, which are burned. The mouth and teeth should be cleansed twice daily with a piece of soft linen and a solution of boric acid. In the intervals between feedings the tongue is kept moist by frequent draughts of water.

Bronchopneumonia.

(*Catarrhal Pneumonia, Lobular Pneumonia.*)

Bronchopneumonia is the prevalent type of pneumonia occurring before the fifth year, but there are also many cases of lobar fibrinous pneumonia during the periods of infancy and early childhood.

Bronchopneumonia occurs both as a primary and a secondary disease. As a primary disease it is most frequent during the first two years of life. Of 605 of my cases of bronchopneumonia, the incidence in regard to age was as follows:

	Cases.
One to three months	32
Three to six months	68
Six to twelve months	207
One to two years	298

These figures correspond within certain limits to those of other authors, although Holt places the greatest frequency between the sixth and the twelfth months.

Sex.—Of the 605 cases, 322 were males—a statement corresponding to that of Jürgensen in regard to lobar pneumonia.

Season.—The greatest frequency is during the winter months, when there are epidemics of influenza during which many primary and secondary cases of bronchopneumonia occur.

Surroundings.—The herding together of the poor certainly has a tendency to increase the prevalence of bronchopneumonia among them. If we believe in the epidemological aspects of pneumonia, it is easy to account for the greater frequency of the disease among the poor: the greater number of their children are rachitic, syphilitic, marantic, and ill-fed, and thus have increased susceptibility to infection.

Secondary bronchopneumonia occurs as a complication in the exanthemata (measles, scarlet fever, typhoid fever), diphtheria, pertussis, and influenza. By far the greater number of cases occur as a sequence of ordinary bronchitis.

Etiology and Bacteriology.—Weichselbaum first demonstrated that the pneumococcus of Fränkel could cause primary bronchopneumonia. His results have been confirmed by Cornil, Babes, and Neumann, the latter of whom found the pneumococcus in cases of primary bronchopneumonia. Quesiner and Neumann found the pneumococcus in the sputum of children suffering from bronchopneumonia.

The secondary form of bronchopneumonia may be caused by streptococci (Northrup and Prudden), which invade the lung-tissue from the trachea, as in diphtheria. Guarnieri also found streptococci in the lungs of children dying with bronchopneumonia after measles. On the other hand, these secondary types of bronchopneumonia may also be caused by the pneumococcus of Fränkel, which causes the primary type of the disease. This has been shown in the work of Netter on the subject, and confirmed by Banti, Strelitz, and Baginsky. In diphtheria the Klebs-Löffler bacillus may be found in the lung areas of secondary bronchopneumonia (Babes, Frosch, Baginsky). The Eberth bacillus has been found in areas of bronchopneumonia complicating typhoid fever (Polyniere).

Morbid Anatomy.—The essential lesion in bronchopneumonia is an inflammation of the walls of the bronchi and of the air-spaces surrounding the inflamed bronchi (Delafield). The walls of the bronchi are thickened and infiltrated with small round cells; those of the alveoli of the lung are thickened and their cavities filled with fibrin, pus, epithelial cells, and new connective tissue. The smaller bronchi are dilated and contain pus, their walls being infiltrated. The inflammation may also be conveyed from the bronchi to the parenchyma of the lung by aspiration of secretion (Ziegler). In the latter case the smaller bronchi are occluded, collapse of the lung follows (atelectasis), and a pneumonia thus results. On section there are seen grayish-red, gray, or yellowish-gray areas of varying consistency, which correspond to a cut bronchus and its surrounding peribronchitic pneumonia. If the areas are croupous, they have a more granular appearance. Small areas of this form of pneumonia may coalesce, and thus whole lobules of the lung be consolidated. These larger areas may be separated by lung-tissue which contains air, or a whole lobe may become consolidated, as in lobar pneumonia. The exudate found in the affected alveoli is at first composed of desquamated swollen epithelial cells, and later of leucocytes. If the exudate has a more fluid character, it is called catarrhal. It then contains more serum than fibrin. If the fibrin is in excess, the exudate has greater consistency, resembling that in lobar pneumonia, and is then called croupous. The catarrhal and croupous forms of exudate may both exist in a lung affected with bronchopneumonia. Blood-cells may predominate in the exudate, so that the lung may on section have a hemorrhagic appearance. This is apt to be the case in streptococcus inflammation and also if foul fluids have been aspirated.

The mucous membrane of the bronchi is the seat of catarrhal inflammation.

There is inflammation of the pleura to a varying degree.

The bronchial and mediastinal lymph-nodes may be enlarged with simple or tuberculous inflammation. There is œdema of the lung tissue which is not inflamed. Bronchopneumonia may result in resolution and restoration to the normal. Suppuration and formation of abscess with destruction of lung tissue, or gangrene of the lung, may result in rare cases.

Persistent bronchopneumonia in children results in induration of the lung. There is an increase of the connective tissue of the alveolar septa, of the walls of the smaller and larger bronchi, and also of the walls of the peribronchial vascular tissue. The lung on section is seen to be studded with fibrous nodules, or a whole lobule or lobe may be converted into connective tissue.

Symptoms.—Bronchopneumonia is divided clinically into several distinct types. In newly born and very young infants the disease may set in insidiously. The infant is born in good condition; after some little exposure it develops slight snuffles and a slight cough. Dyspnœa then appears. All this may occur within the first eight days after birth. The cough becomes more harassing and the dyspnœa more marked. Slight cyanosis supervenes after a time. The infant is restless and does not sleep, the cyanosis becoming more marked and constant. The infant may have frequent convulsions. The dyspnœa finally becomes so marked as to cause distinct drawing inward of the lower part of the chest-wall with each inspiration. In these cases there is little or no temperature; in that respect they resemble cases of bronchopneumonia in extremely old people. The temperature may be slightly subnormal even when the infant is mortally ill with a disseminated bronchopneumonia. The cough may not be marked. These cases should be differentiated from those occurring in infants born with an atelectatic condition of the lungs. In the class of cases under consideration, atelectasis develops as a sequence of the bronchitis and bronchopneumonia. The movements are greenish, containing undigested curds. The infants may finally develop enteritis. The course of the disease is in these cases very acute. The infant either rapidly grows worse or begins to improve immediately. The former course is, however, the rule in this very dangerous and insidious form of bronchopneumonia. If the infant does not improve, the cyanosis becomes more marked, as does also the dyspnœa; the respirations increase to more than 80 a minute, the pulse becomes very rapid, and the heart feeble; the infant lies in a soporose state; the end may supervene with tympanites, convulsions, and œdema of the lung. This form of bronchopneumonia is very frequently overlooked at the outset and mistaken for a simple bronchitis.

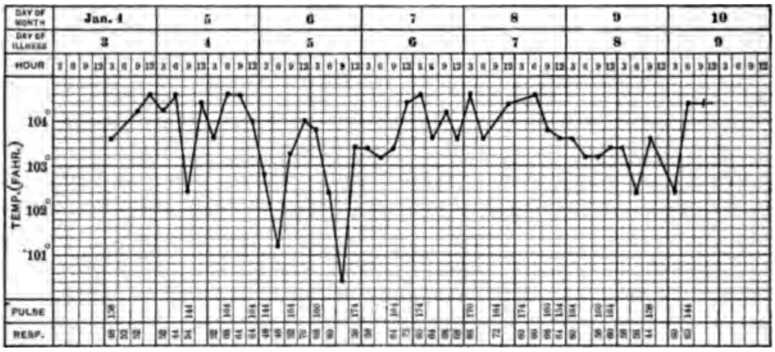
Another form of bronchopneumonia in infancy begins as a simple bronchitis, and may be treated as such for days. Finally, posteriorly in both lungs there are found the fine crepitations which give warning of the presence of bronchopneumonic processes. Bronchopneumonia of this variety runs its course without temperature. It occurs in rachitic or weakly infants and children, or follows a mild attack of influenza. The attacks of coughing are especially troublesome, and are frequently followed by vomiting of the contents of the stomach. The movements are loose, and show greenish particles and undigested white flaky masses. The dyspnœa is constant and characteristic, and if the patient is out of bed, grows more marked toward the late afternoon. The alæ nasi are dilated. The temperature rarely rises above 101° F. (38.3° C.), and is generally 100° F. (37.2° C.) or even lower. The course is favorable; the cough may persist for weeks after the subsidence of the acute symptoms, being especially marked at night.

A more common form of bronchopneumonia in infancy begins as a simple bronchitis, which may last for a few days, when, without warning, the infant has a chill followed by a rise of temperature, the case having suddenly developed into a full bronchopneumonia. In a six weeks' old infant with disseminated patches of pneumonia, the chill was so severe as to cause extravasations of blood underneath the surface, with markings resembling those seen in marbling of the surface. In another case the chill was so severe that an immediate fatal issue was feared. In that bronchopneumonia sometimes begins with a chill, it resembles a lobar process.

The most common type of bronchopneumonia may begin with a rise of temperature preceded by vomiting. The harassing cough is present from the outset, causing the patients to cry with pain at each attack. There is no sputum, but in very young infants a frothy mucus may in the later stages of the disease collect about the mouth. The dyspnœa is marked. The alæ nasi are dilated at each inspiratory effort. The peripneumonic groove is depressed, and in very severe dyspnœa the suprasternal region may also be depressed at each inspiration. Very frequently the dyspnœa will resemble that due to laryngeal stenosis. There are, however, none of the signs of laryngeal obstruction, such as laryngeal breathing.

Fever is always present in infants and children, except in the classes of cases above noted. It may reach 106° F. (41.1° C.), and is as a rule remittent. It may fall gradually to the normal, and in the favorable cases may reach the subnormal and remain there for a few days. The course of the fever is, however, not an indication of the severity of the disease. Fatal bronchopneumonia sometimes shows a steady decline in the temperature toward the approach of the fatal issue. In other cases the temperature may drop to the normal, remain there a few hours or a day, and then rise sharply to 104° F. (40° C.) or higher, thus indicating that a new part of the lung has been invaded by the disease (Fig. 114). Such rises of temperature after a fall to the normal are of grave import if they occur in an infant acutely ill with a process which has been severe for days. They show a tendency of the process to spread, and in young weakly infants such an extension of the process is apt to be fatal. A drop by lysis to a normal temperature which con-

FIG. 114.

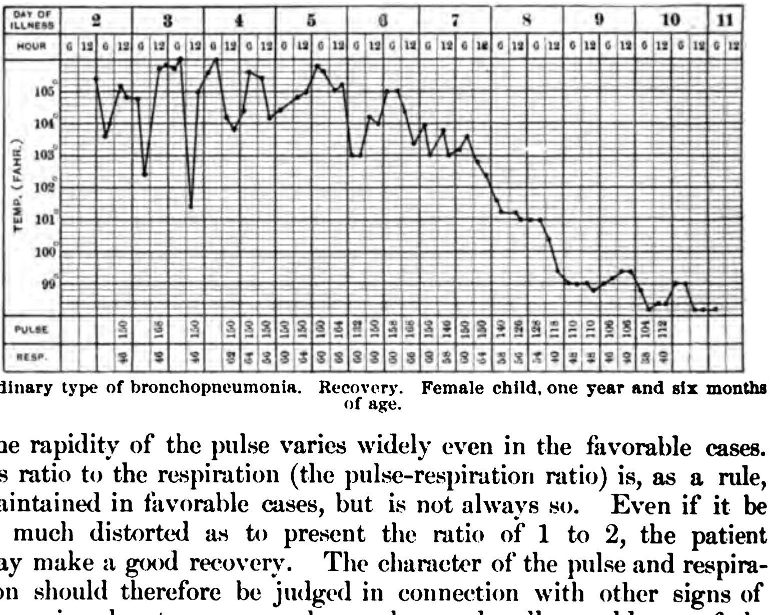


Fatal bronchopneumonia; reinvasion of the lung on the fifth day. Infant, six months old.

tinues for a few days, and is followed by a slight gradual rise with subsequent remissions to the normal is also common, and may indicate a return of the bronchopneumonic process, or a pleuritic effusion of a purulent character. The physician should be on the alert for an effusion in the cases which have run an irregular or remittent temperature for a period of more than two weeks. I have, however, operated upon cases of empyema following bronchopneumonia in infants, in which the temperature-curve was normal for days, and then showed occasional rises to 101° or 102° F. (38.3° or 38.8° C.).

The **pulse** is as a rule rapid. It is difficult in infants to estimate its exact character. It is, however, always possible to distinguish the abnormally weak and thready pulse even in the youngest infant

FIG. 115.



Ordinary type of bronchopneumonia. Recovery. Female child, one year and six months of age.

The rapidity of the pulse varies widely even in the favorable cases. Its ratio to the respiration (the pulse-respiration ratio) is, as a rule, maintained in favorable cases, but is not always so. Even if it be so much distorted as to present the ratio of 1 to 2, the patient may make a good recovery. The character of the pulse and respiration should therefore be judged in connection with other signs of decreasing heart power, such as abnormal pallor, coldness of the surface, and cyanosis however slight. In artificially-fed infants who are above the average weight, the beginning of cardiac weakness is indicated by an abnormal pallor of the face and slight cyanosis of the lips.

Sputum.—In young infants there is no sputum, nor is it probable that in uncomplicated cases of bronchopneumonia the younger infants cough up and swallow sputum, as is generally supposed. At most, there is after severe attacks of coughing a collection about the lips of frothy mucus probably coming from the trachea.

Gastro-enteric Tract.—The symptoms referable to the stomach and intestine are of great importance in severe bronchopneumonia of the primary type. Even up to the second year of life tympanites sets in very early. In one case it was so distressing a symptom as to mislead the physician into thinking that peritonitis might be present. It is especially apt to set in with rachitic and weakly infants who have been fed on the bottle. If it appears late in a very sick infant, it is a symptom of grave import, and may sometimes cause the fatal issue. In some cases the pre-agonal distention is very extreme, and so far as can be judged very painful. Some infants begin to vomit from the outset of the pneumonia. The vomiting may occur once or twice in the twenty-four hours, or may be incessant. With the vomiting there may be the passage of greenish stools or a fully developed enteritis of severe type. So severe is the enteritis in some cases as to cause the death of a patient suffering from pneumonia of only moderate severity. This form of the disease does not occur exclusively in the summer months, but is more prevalent at that time.

Cerebral Symptoms.—The infant is in some cases stupid from the outset of the disease. Older children may have slight convulsive twitchings of the muscles of the face and extremities. In cases in children at the third year there may be complete unconsciousness and symptoms simulating those of meningitis, such as rigidity of the muscles of the neck. I have seen the cerebral symptoms persist for weeks in young infants who made complete recoveries. In other cases in young infants and children, the bronchopneumonia may partly resolve, and still there may be a continuance of the cerebral symptoms or even an exacerbation of them. In these cases the possibility of the presence of otitis or mastoid inflammation should be seriously considered.

The secondary form of bronchopneumonia may complicate the exanthemata—measles, scarlet fever, varicella, typhoid fever, pertussis, influenza, and diphtheria, and also gastro-enteritis or any form of infection, such as that of septic wounds or osteomyelitis.

The symptoms of bronchopneumonia which complicates **pertussis** are of an unequivocal character. A febrile movement may be present with a simple bronchitis. If bronchopneumonia is imminent or present, the fever is marked and constant, and may reach 106° F. (41.1° C.). The dyspnœa is very marked, but the cough may not be increased. In certain forms of pertussis without complications there is a slight constant dyspnœa, which is due to the disease. If bronchopneumonia is a complication the dyspnœa is more decided, the number of respirations three or four times the normal, and the pulse-rate increased. There is marked cyanosis. There may be all the symptoms of a severe bronchopneumonia, such as tympanites, vomiting, and green diarrhœal stools. The bronchopneumonia is, as a rule, of the disseminated type, with areas of consolidation of greater or lesser extent in both lungs. The infants are much more ill than they would be with a primary process of the same extent. A bronchopneumonia of this kind can be diagnosed if upon examination of the chest there are, in addition to the physical signs of bronchitis, fine crepitations over the different parts of the chest, especially over the lower lobes of both lungs posteriorly. There may also be dulness with bronchophony and bronchial breathing over small areas, either in the upper or lower lobes of the lung on one or both sides. The bronchopneumonia of pertussis may supervene at any period of the disease, and is not the result of exposure. On the contrary, it may occur in infants and children who have been most carefully protected from exposure. It is the result of the form of disease—a mixed infection. The pertussis probably makes the lung more viable to disease in some subjects than in others. The bronchopneumonia is a grave complication, and is very fatal. It may cause complications, such as pleurisy of a serous or purulent nature, and often opens the way for invasion of the lung by tuberculosis. It may run a chronic course (persistent pneumonia) and reduce the patient to a very weak state. The patient will then develop consolidation of a whole lobe of the lung which will take weeks to clear up.

Bronchopneumonia complicating **measles** supervenes, as a rule, in the stage of eruption, and is a very serious complication. Its presence may be suspected if, on examination, of the chest, there are found, in addition to the râles of bronchitis, very fine crepitant râles over areas disseminated through both lungs. This complication also causes a febrile movement after the fading of the eruption. There are severe cough and dyspnœa. The pulse may reach 180 to 190, and the respirations 90, but the patient may recover even if the signs of cardiac weakness, such as cyanosis, are marked. The patient is stupid, does not take food or notice his surroundings. Sometimes there may be other signs, such as hemorrhages into the eruption (so-called hemorrhagic measles), indicating that the process is one in which there is a mixed infection. There may be a complication of serous or seropurulent pleurisy.

Bronchopneumonia complicating **typhoid fever** does not, as a rule, give very striking features apart from those belonging to the latter disease. It seems to be of a mild and insidious character. The bronchopneumonia of typhoid fever is apt to mask the typhoid if it appears at the outset of the disease. There is then a typhoid beginning as a pneumonia. The area of bronchopneumonia is well localized. It may be a small area in the upper or mid-region of the lung. The febrile curve in these cases may range quite high at the outset and thus mislead the physician. The process persists for weeks, sometimes as long as five weeks. The lung is slow in clearing up. The signs of dulness, bronchial voice and breathing may persist into convalescence. In other cases the pneumonia may supervene in the course of the disease. It can then be detected only if the cough is harassing and the dyspnœa marked. In delirious patients the pneumonia can only be discovered by repeated and constant examination of the chest. These cases are not so apt to develop pleurisy of a serous or purulent nature as the pneumonia complicating measles or scarlet fever.

Varicella is only rarely complicated by bronchopneumonia. In this disease also the pneumonia runs a protracted course, but is less serious in its outcome than in the other exanthemata. It occurs in the severer forms of varicella in which the eruption is complicated with abscesses or necrosis of the skin (mixed infection).

Scarlet fever is not so frequently complicated by bronchopneumonia as measles, but when it does occur the bronchopneumonia is of a very severe type. It occurs in the septic forms of scarlet fever, and may appear early in the disease, on the fading of the eruption. Scarlet fever complicated by bronchopneumonia is frequently followed by pleurisy of a purulent nature.

The bronchopneumonia which complicates **diphtheria** has been carefully studied by Northrup and Prudden. It is the result of a streptococcic invasion of the lung or an invasion by the Klebs-Löffler bacillus. As a rule, however, it is a mixed infection, as was pointed out by Northrup and Prudden. The laryngeal form of diphtheria frequently proves fatal through this complication.

Of special interest is the bronchopneumonia which complicates chronic or subacute **diarrhœal conditions.** This form, which is of a distinctly septic type, is caused by infection of the lung by streptococci, which invade the general circulation through erosions in the mucous membrane of the gut (Booker, Czerny, Fischl). It is not always due, as was formerly supposed, to keeping the infant in the recumbent posture, nor does it occur in hospital practice alone, but is frequently seen in private practice in infants in unhygienic surroundings. It is of the persistent type, and runs its course with a daily high or low febrile curve, and results in areas of consolidation, which sometimes involve a whole lobe of a lung. This form of pneumonia is one of the fatal complications of the subacute intestinal catarrhs.

Some infants, after one attack of bronchopneumonia, have repeated or recurrent attacks on the least exposure (Fig. 116), in some cases developing catarrhal croup. In other cases, there develops an emphysematous condition of the lung, in which the least exposure or change in the atmosphere will cause an asthmatic attack.

Course, Termination, and Complications.—Bronchopneumonia may terminate in complete recovery and restoration of the lung to the normal, or may prove fatal. The mortality varies at different times and with the environment. The prognosis in marantic infants, and also in bottle-fed infants, is very bad. Rachitic infants have bronchopneumonia with a very protracted course (Fig. 117). The forms which complicate measles, pertussis, scarlet fever, and influenza are very fatal. Abscess or gangrene of the lung may be a complication. In some forms of otitis the symptoms may very closely simulate those of tuberculous meningitis. Otitis prolongs the disease and frequently misleads the physician. Especially trying are the forms of bronchopneumonia of very limited extent in one or both lungs, in which, after the disease has run its course, there is a protracted, remittent or intermittent fever-curve. Serous pleurisy and empyema are very common complications. Their presence may be suspected if the disease runs a course protracted beyond two weeks, and if signs, such as dulness, flatness, and bronchophony, persist and become more marked over the whole side of the chest.

Meningitis may complicate the disease. Care should be taken not to confound cerebral symptoms with true meningitis.

Pericarditis complicating bronchopneumonia is apt to be purulent, and is rarely diagnosed during life. I have seen cases in which during life repeated examinations failed to reveal positive signs of effusion into the pericardium, but in which purulent pericarditis was found at autopsy. This is frequently true of cases in which the effusion is limited (30–50 grammes). If bronchopneumonia occurs in the left lung with consolidation anteriorly and some pleural effusion, it is almost impossible to diagnose moderate pericardial effusion. The complication is very fatal.

Pfisterer has recently published a number of cases of pneumococcus osteomyelitis and metastases occurring by way of the blood or lymph-stream. In some cases the arthritis may precede the pneumonia; in others, may follow it. The portals of infection include the tonsil, among others the mouth or nose, the ear or peritoneum. Traumatism may be a predisposing factor. Netter found 3 of arthritis in 1218 cases of pneumonia. It is therefore rare as compared to other complications of pneumonia, such as otitis or meningitis. I have seen one case in a newborn infant, the subject of congenital syphilis, with bronchopneumonia of a syphilitic character. In this case the hip-joint was the seat of pneumococcus suppuration. As a rule, the larger joints, the shoulder or knee, are affected. It is generally monarticular, but may be polyarticular. The symptoms in some cases escape observation; in others, the symptoms are similar to those of osteomyelitis with arthritis. If the arthritis is very acute and other organs are involved, death may result; but, on the other hand, if the joint is evacuated in time, recovery may take place. The pneumococcus arthritis involves the tissues surrounding the joints. The cartilages of the joint are rarely involved. Pneumococcus osteitis affects the cortical layers of the bone in the vicinity

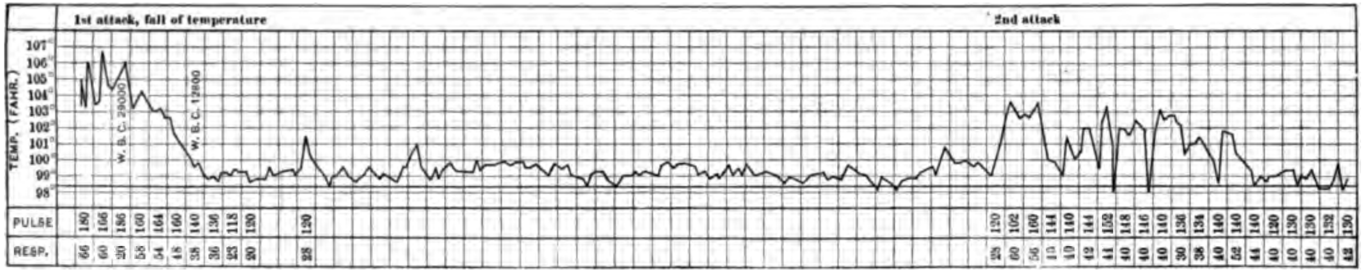


FIG. 116.—Recurrent bronchopneumonia. Infant, nine months of age. First attack involved the lower lobe of the left lung and an area in the right lung. Recurrence in the right lung, lower lobe. Recovery.

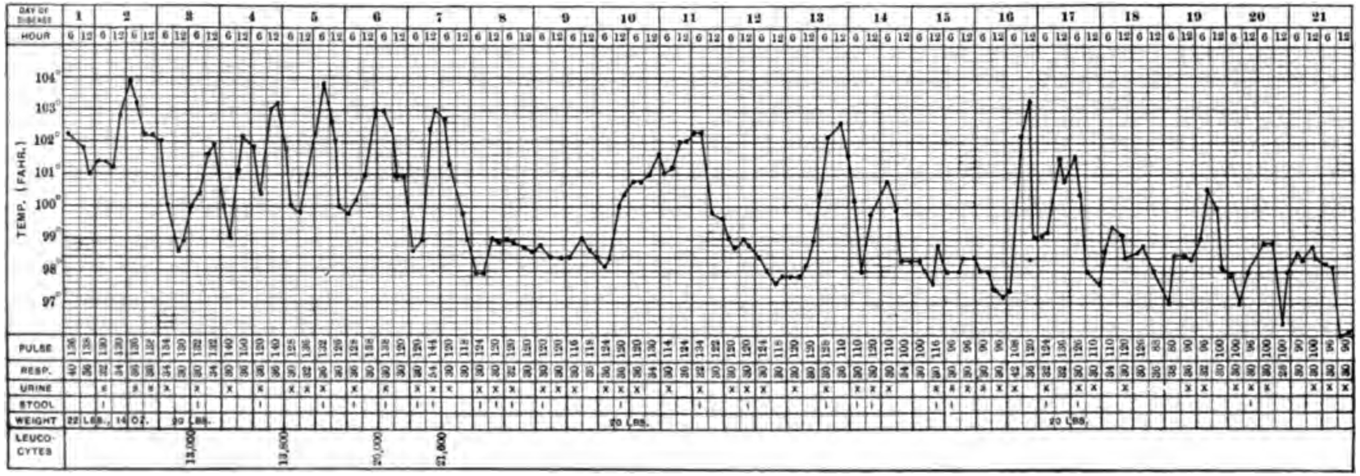


FIG. 117.—Bronchopneumonia of prolonged course, showing rises of temperature due to the invasion of a new area of the lung. Female child, two years of age.

of the epiphyseal line. Large sequestra are rare. Of 41 cases, 15 occurred in childhood in the first two years of life.

The **prognosis** is good, according to some; but of 44 cases of pneumococcus arthritis and osteitis collected by Pfisterer, 23 died. Death was due to complications, such as pneumonia, empyema, meningitis, endo- or pericarditis; therefore the prognosis is good only in uncomplicated cases.

Physical Signs.—Clinically the physical signs of bronchopneumonia are divided in those of the following stages: the first stage—invasion, the second stage—consolidation, the third stage—resolution. There is no sharp line of demarcation between the signs of the stages.

First Stage.—Inspection shows the face to be flushed on one or both sides, and the nostrils to be dilated; with each inspiration there is drawing inward of the peripneumonic groove and sometimes of the suprasternal tissues over the upper part of the trachea.

PALPATION.—If bronchitis is present, there may be rhonchal fremitus, but it is frequently absent.

PERCUSSION.—In the early stage, there is, just before consolidation, slight dulness over small areas, which in young infants with thin-walled chests may have a slightly tympanitic note (tympanitic dulness). Other parts of the chest may have a vesiculotympanitic note.

AUSCULTATION.—If bronchitis is present, the râles of bronchitis may be heard. The respiratory murmur is rude. By careful examination of all parts of the chest one or more areas in which are heard fine crepitant râles may be found. They may easily be overlooked, and may disappear when the infant cries or coughs, and during the examination.

VOCAL RESONANCE is slightly increased over areas in which there is slight dulness or the beginning of consolidation. The whole posterior aspect of the thorax from above downward, and also the axillary fossa, should be examined. The apex of the lung in front, and the lower part of the thorax in front and behind on both sides, should be carefully examined, as well as the areas of the borders of the lungs where they come in contact with the chest-wall. Increased vocal resonance and slight dulness alone, especially over the apex of the right lung in front and behind, should be accepted with great caution as indicative of the beginning of bronchopneumonia.

Dyspnœa should not be looked upon as a sign of pneumonia. The crepitant râle in a circumscribed area or in several areas is the sign pathognomonic of this stage.

Second Stage.—INSPECTION shows no condition differing from those of the first stage.

PALPATION.—If the area of consolidation is limited, there is no change, because the area and the chest are small. If there is effusion in the lower portion of the pleural cavity, the fremitus may be diminished over the lower part of the chest, although the pneumonia is in the upper part. Fremitus is therefore misleading, and is only confirmatory in the presence of other signs.

PERCUSSION reveals dulness in complete consolidation or dulness with a tympanitic note in the beginning of consolidation, and also flatness if fluid is present over the consolidated area in the lower part of the lung. The dulness may involve a very small area or an entire lobe of the lung. There may be slight resistance to the percussing finger over the consolidated area. The unaffected lung is hyperresonant.

AUSCULTATION gives bronchophony and bronchial or bronchovesicular breathing over the consolidated areas. These are not necessarily present over consolidated lung. In infants and children there may only be abnormally rude respiratory murmur and increased vocal resonance. Fine crepitant pleuritic râles may be heard over the consolidated area.

Diagnostic stress is to be laid on complete dulness with bronchophony and bronchial breathing.

Third Stage.—PALPATION will give increased fremitus if the area of lung consolidation is large and there is no fluid over the area.

PERCUSSION.—As in the first stage, there is dulness to a varying extent, with a tympanitic note showing the return of air into the lung.

AUSCULTATION gives a crepitant râle, as in lobar pneumonia. The voice and breathing are less bronchophonic. Dulness may persist for days or weeks. In some cases there is fluid, which increases the dulness or flatness. Dulness, crepitant râles, bronchophony, and bronchial breathing are constant features, and are diagnostic. In infants and children, bronchophony is more constantly present than bronchial breathing. In the bronchopneumonia of the newly born infant, it is sometimes possible to discover with the small bell of a stethoscope areas in which air does not enter (atelectatic).

Equivocal Signs likely to be Mistaken for the Beginning of Bronchopneumonia.—In infants and children, the physician is apt to be easily misled into a diagnosis of incipient bronchopneumonia. Equivocal signs—*i. e.*, signs which are not absolutely diagnostic—are apt to be met in certain parts of the chest and in the presence of rational symptoms, such as fever or apparent dyspnœa, undue importance may be attached to them. These signs are as follows:

a. A slightly high note on percussion and an increase of vocal resonance or fremitus, with a rude respiratory murmur on the right side over the apex in front or behind. It should not be forgotten that this region, especially in infants, normally shows varying degrees of these signs as compared with the left side.

b. A slight dulness over the lower part of the chest on the right side behind, due to the presence of the liver, is normal. To be abnormal, the dulness must be very marked and the vocal resonance much increased. The resistance to percussion must be pronounced in order, in the absence of more positive signs, to justify a suspicion of the beginning of consolidation.

c. Bronchial or bronchovesicular breathing too near the vertebral column behind on either side, between the scapulæ, should be cautiously interpreted. In some infants, the breathing in this region is normally bronchovesicular. It is in this region that the diagnosis of *central pneumonia* is so often made—a diagnosis rarely verified by the subsequent course of a case.

d. In some infants and children, especially from six to ten years of age, it is found that the fremitus and vocal resonance diminish behind from a short distance below the angle of the scapula to the base of the lung; the breathing also is heard less distinctly. A diagnosis of incipient pneumonia or consolidation with fluid requires positive and unmistakable evidence very low down behind. The thick muscles of the back and organs behind the thorax, such as the kidney and liver, obscure slight signs below the ninth or tenth rib. Slight variations from the normal should not receive undue attention.

Diagnosis.—Bronchopneumonia should be differentiated from the lobar fibrinous form of the disease. In children above five years of age this is not difficult; in those under the second year, in whom fibrinous or lobar pneumonia is not uncommon, a positive diagnosis of lobar pneumonia cannot be made until the stage of consolidation, and even at that time only as to distribution. In the main, it is made from the course of the temperature. In lobar pneumonia the temperature will fall by crisis after the usual period. A marked leucocytosis, which increases toward the day of crisis and then rapidly diminishes, is also a characteristic feature. There should be also the physical signs of lobar consolidation.

If these symptoms and signs are all present, it may be assumed *clinically* that a lobar pneumonia is to be dealt with. Such a diagnosis is always open to doubt, for a bronchopneumonia may have the lobar consolidation and the leucocytosis, but will rarely have the critical drop of temperature which occurs in lobar pneumonia. As to the onset, bronchopneumonia may set in with a chill, and lobar without one. The complications in both forms are identical; empyema is as likely to occur in one as in the other. Lobar pneumonia is rarely prolonged in duration if complications are absent, while the bronchopneumonic type of disease is, as a rule, of longer duration and may be prolonged into a chronic course.

Disseminated patches of consolidation in a lung in which there is general bronchitis point to bronchopneumonia; diffuse bronchitis.

with fine crepitations in the lower lobes of both lungs, to bronchopneumonia. The presence of a primary disease—measles, scarlet fever, typhoid fever, and influenza—will also influence the process in the lung. The secondary pneumonia is a bronchopneumonic process.

Prognosis.—The mortality of bronchopneumonia, even under the favorable conditions of private practice, is as high as 25 per cent. In hospital practice it is much higher, and may reach 50 per cent. or more. It is increased in bottle-fed, rachitic, prematurely born, and syphilitic infants, and is greatest in the first year of life. The disease is especially fatal in newly-born infants, and in cases of gastro-intestinal disorder. The mortality rate increases in New York City in the months of December, January, and February, during which the weather is alternately moist, warm, and cold. Certain years show an increased mortality because of the severe nature of the epidemic.

At the bedside, a prognosis is based on the condition of the lung, temperature, heart, and the presence or absence of nervous symptoms. A persistently high temperature, if there are areas of consolidation in both lungs, is of serious import. An abnormal pallor or slight cyanosis in a bottle-fed baby, even if well-nourished, is a danger signal. Forced and irregular action of the diaphragm is serious; marked drawing inward of the sides of the chest, sometimes as high as the eighth rib, is a very unfavorable sign in infants. These cases show a depression of the suprasternal notch as marked as that which occurs in laryngeal obstruction. Repeated convulsions and jaundice, with enlargement of the spleen, in rachitic infants indicate intense toxæmia. These cases are fatal. Marked tympanites at the end of the first week, in connection with diarrhœa and weakness of the heart, is an unfavorable symptom. Dyspnœa with respirations irregular in rhythm and depth denotes diffuse involvement of both lungs, and is present in the unfavorable cases. Cerebral symptoms supervening late in the disease are unfavorable.

The favorable signs are a good muscular quality of the first sound of the heart, red lips and warm surface, good reaction after hydrotherapy, and periods of quiet sleep with full noiseless breathing, movements of the bowels normal or slightly green, and an absence of marked tympanites. Caution should be exercised in making any prognosis in a bronchopneumonia which shows a marked tendency to involve new areas of lung.

In the **treatment** of bronchopneumonia of infants and children, it should be borne in mind that the disease is a self-limited, acute, infectious one, and that there is no remedy which can abort it or prevent complications. As in lobar pneumonia, the ill effects of the disease must be counteracted as much as possible and the strength of the patient supported. Since the patients are of very tender age, remedies which are powerful in their ultimate effects are to be carefully avoided. The indications in the treatment are to counteract the effects of the temperature and to support the heart.

The **temperature** in the most fatal forms of this disease in newborn infants is below the normal at times, and rarely reaches a very high point. In other cases of bronchopneumonia in older infants and children, it remains persistently above 103° F. (39.7° C.). In these cases, as in lobar pneumonia, the various forms of hydrotherapy are utilized. Of all the methods, the cold compress applied to the chest, as before described, seems to be the most efficacious. Compresses lower than 70° F. (21.1° C.) are not applied. The applications may be renewed every hour, if the patient bears them well. It sometimes happens that a compress wrung out in water at 70° F. (21.1° C.) will depress the patient, causing cyanosis wthout reaction. In such cases, as in the lobar cases, I have found the warm bath, 105°–107° (40.3°–41.6° C.), of the greatest utility in relieving the nervous symptoms, such as restlessness and convulsive twitchings. Infants, as a rule, will not bear baths below 80° F. (26.6° C.). I therefore do not utilize the cold full bath in bronchopneumonia in infants. I do not think it advisable to use the bath at 90° F. (32.2° C.) or higher, with cold douching of the head and shoulders, to obtain reaction in infants. The procedure rouses the patients only momentarily, and the subsequent depression is greater. Cold packs over the whole body are also heroic remedies, but are advocated by some authors.

The **heart** is supported by means of digitalis, strychnine, camphor, musk, caffeine, and ammonium carbonate. Of these agents, the most useful are digitalis, strychnine, and musk.

Digitalis is administered in the form of the tincture. A drop is given for every six months of the age of the patient. It should not be used unless the pulse is high, and should then be given every three hours. It is discontinued after being administered for two or three days. The effects of stronger preparations, such as the fluid extract, cannot be gauged so carefully as those of the tincture, and they are therefore less useful. The cases in which digitalis is of the greatest value are those in which there is cyanosis to a mild degree, or excessive pallor denoting great cardiac weakness.

Strophanthus may be administered alone or in combination with digitalis. The tincture is the form generally used.

Strychnine is one of the most useful drugs in the treatment of the heart. An infant six months old will bear grain $\frac{1}{250}$ or $\frac{1}{200}$ (0.0003 or 0.00025) very well. Older infants and children bear grain $\frac{1}{150}$ (0.0004) quite well. Strychnine should not be used in cases where there is excitability of the nervous system.

Atropine, which is so useful in adults, is not well borne by infants and children.

Ammonium carbonate is one of the most useful drugs when for any reason digitalis cannot be used. Convulsions or restlessness are treated with the bromides of potassium and sodium, which may be combined. Chloral hydrate is combined with both, especially where one dose of bromide of potassium and chloral hydrate is given per rectum.

I do not use poultices. Some authors use them as a routine measure.

Inhalations of benzoin and turpentine are of little efficacy. They do not affect the local lesion in the lung, nor do they act on the mucous membrane as they do in catarrhal processes of the nose and throat. In some cases I have seen harm result from overloading the atmosphere with the odor of balsams.

The patient should be isolated from the healthy children of the family and the room kept at a temperature of from 68° to 70° F. (20° to 21.1° C.) and well ventilated. An open wood fire is the most satisfactory method of heating and ventilating the sick-room.

In threatened œdema of the lungs I have found, as in lobar pneumonia, that the right ventricle is best relieved by nitroglycerin, grain $\frac{1}{200}$ to $\frac{1}{100}$ (0.0003 to 0.006) at a dose, and by the constant administration of oxygen containing 20 per cent. of nitrous oxide.

Whiskey is so universally used that the mode of administering it should receive special mention. Alcohol should not be used as a routine remedy. In some of the milder cases its use is superfluous. There are other cases in which its use must be suspended because of the constant vomiting. In the severer types of bronchopneumonia, in which the temperature is persistently high, the effects of the toxæmia may be counteracted by administering whiskey. Infants receive from minims xx to xxx (1.2 to 2.0); older children a drachm (4.0) every three hours. The whiskey should be well diluted, and should be given after the nursings.

The feeding of infants who take a substitute for the breast should be carefully watched, especially in bronchopneumonia, a disease in which diarrhœa is apt to supervene. If diarrhœa is present, the milk should be discontinued and a cathartic given. The infant is given a high rectal injection of warm normal saline solution twice daily, and is kept on solutions of egg-albumin and acorn cocoa until the intestinal symptoms subside. Milk is then again given. In these cases of intestinal disorder it is of the utmost importance to see that the milk is fresh and uncontaminated.

The cases not complicated by diarrhœa are given a warm high rectal enema of the normal saline solution once daily. In infants, this procedure will ward off tympanitic distention of the abdomen and stimulate the heart.

The cough is sometimes very harassing, and then only should be relieved. The camphorated tincture of opium or the wine may be given in moderate doses. Codeine is useful in older children; morphine should not be used. In the many hundreds of cases which I have treated I have not found it necessary to use it. Strapping the chest to relieve pain is harmful in infants and children. The chest in these subjects is resilient, and any limitation of its motion reacts unfavorably in preventing a full expansion of the unaffected lung.

Persistent Bronchopneumonia.

(*Chronic Bronchopneumonia.*)

Persistent bronchopneumonia is a distinct type of bronchopneumonia the course of which extends over weeks or months, the patient meanwhile becoming much reduced in flesh and strength. These cases occur in weakly infants, usually in those who are bottle-fed. A distinct type of the disease complicates chronic enteric catarrh. Cases of this class belong in the category of Gastro-Intestinal Sepsis of Fischl, Escherich, and Czerny. Cases of another set complicate and follow pertussis, measles, and influenza. Lastly, there is a true tuberculous form which is not strictly included in the above classification. The condition is thus rarely primary.

Symptoms.—The infant or child has at first the symptoms of an ordinary bronchopneumonia. The fever, however, is of longer duration than in cases which recover. Cases of gastro-enteric affection or pertussis will continue to have a remittently high temperature, which may reach 105° (40.5° C.), but fall to 101° or 100° (38.3° or 37.7° C.) on the same day. It will remain normal for days, and then rise again, as indicated in the chart (Fig. 118). There are cough, slight dyspnœa, emaciation, and gastro-intestinal disturbances. In cases of enteric catarrh the intestinal disease takes clinically a secondary place. Some of these cases eventually recover in spite of the progressive emaciation and high fever. This is especially the case in persistent bronchopneumonia which complicates pertussis.

The Blood in Persistent Bronchopneumonia with Recurrent Invasions.—In the case from which the chart was taken there was a distinct increase of the number of leucocytes with each new rise of temperature and fresh invasion of the lung. The number of leucocytes mounted as high as 80,000 to the cubic millimetre. A differential count showed that the polynuclear neutrophiles ranged at different times from 73 to 82 per cent. of the leucocytes and the small lymphocytes (mononuclear) from 13 to 21 per cent. As the disease progressed, there were also signs of extreme anæmia, microcytes, megalocytes, and poikilocytes being present.

Physical Signs.—On examination, there are found areas of consolidation of varying extent, generally made out posteriorly over the apex or toward the base of the lung. There are signs of general bronchitis, increase of fremitus, and dulness marked, slight, or combined with a tympanitic note. There may be fine crepitations here and there over the chest. If the areas are extensive, there may be bronchophony or bronchial breathing. The complete consolidation of primary bronchopneumonia is not always present. The lung is only partially consolidated, so that the vocal resonance may simply be markedly increased or the breathing may be bronchovesicular.

Diagnosis.—Persistent bronchopneumonia may be suspected if there is an area of dulness at the apex of one lung which does not resolve after a lapse of weeks. In these cases, there are the other signs of partial or complete consolidation at the apices, the base of the lung continuing resonant in the absence of signs of pleurisy. The persistence of fremitus on the affected side, especially in the midregion of the chest behind, will aid in excluding the presence of fluid. The rest of the lung is in these cases resonant or hyperresonant. In doubtful cases the exploring-needle should be introduced into the chest to ascertain whether fluid is present.

The **treatment** is practically an extension of the treatment of the primary condition. If there is an affection of the gastro-enteric tract,

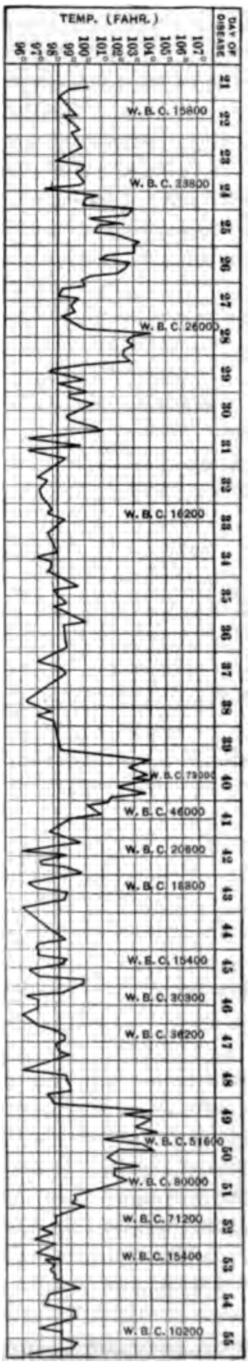


FIG. 118.

Persistent bronchopneumonia with areas n the left and the right lung. With each new invasion of the lung there is a marked leucocytosis, which disappears as the temperature falls to the normal. Recovery. Female child, sixteen months of age.

it is treated. If there is pertussis, treatment proceeds on the lines usually followed in that affection. In some cases the administration of iodide of potassium in small doses has seemed to have a beneficial effect on the course of the process in the lung.

VI. DISEASES OF THE PLEURA

Pleurisy.

(*Pleuritis.*)

Pleurisy in infancy usually occurs as a secondary disease; it is rarely primary.

Dry pleurisy is the form in which the pleura is inflamed without any appreciable formation of exudate in the pleural cavity.

Pleurisy with effusion, or subacute pleurisy, as it is incorrectly called, is the form in which a serous or serofibrinous effusion is found in the pleural cavity. The form in which the effusion is of a seropurulent or markedly purulent character is also called empyema.

Empyema is therefore a purulent or suppurative pleurisy. There are other forms of pleurisy which occur with neoplasms of the lung or pleuræ. These are not discussed in this section.

Dry Pleurisy.

Frequency.—Dry pleurisy, pure and simple, is, in my experience, clinically not common among infants and young children. As an independent affection, it is found more frequently after the fifth year of life. Clinically, the cause of this infrequency in infancy cannot be easily explained. Young infants and children rarely indicate the pain which is the leading symptom. The disease is masked by other symptoms occurring at the same time. Older children locate the pain and direct attention to it.

Etiology.—This form may be primary or secondary. As a primary affection it is found in rheumatic subjects, especially those who are or have been subjects of disorders such as endocarditis or fibrinous adhesive pericarditis. In these cases the etiology is the same as that of rheumatism. The condition is secondary to pneumonia. It may be found complicating any of the infectious diseases—influenza, scarlet fever, measles, typhoid fever, or tuberculosis. In such cases the bacterial factor in the etiology is much the same as in the forms which will be considered under Pleurisy with Effusion. Pleurisy may complicate nephritis of the subacute or chronic type. Traumatism will cause this form of pleurisy; exposure to cold or wet will predispose to it.

Symptoms.—The cases of simple dry pleurisy not proceeding to the formation of effusion in the pleura, which have come under my notice, gave few symptoms.

Pain.—The children in the majority of cases complained of distinct localized pain on exertion or on breathing deeply. There is also some local pain on external pressure. I have seen marked pleurisy of the dry form in which pain was absent. This is most likely to occur in pleurisies secondary to nephritis. In the primary type, the patients continue to walk about, but are pale and have an anxious expression of the face. There is sometimes a rise of a degree or more in temperature and the respirations are increased and superficial. Those forms described by Henoch as setting in with convulsions, high fever, and vomiting, have not in my experience remained dry fibrinous pleurisy, but have proceeded to the formation of effusion in the chest. The duration of dry pleurisy is variable, and in the rheumatic forms may extend over a long period of time.

The **diagnosis** is not difficult, and is made from the physical signs and the history. On examination, a localized area over which there are a large number of dry crepitant râles is found. The râles are heard so close under the ear that they are distinguishable from the crepitant râles of pneumonia. In some cases there is a dry rubbing sound—a pleuritic friction—over the area affected. In the cases without complications there are no other signs. There is little or no dulness and no change in the voice or breathing-sounds.

The **prognosis** is very good. Tuberculous disease of the lung is not a causative agent in these cases in children so frequently as in the adult. The primary dry pleurisies, with proper care, subside and gradually disappear.

The **treatment** of dry pleurisy is very simple. If the subjects are rheumatic, they are put on small doses of salicylate of sodium. The bowels are kept open with a saline cathartic, preferably Carlsbad salts. The patients are kept in bed. It is not advisable to strap the chest to relieve pain. The desired relief can be secured by some local application of iodine or a sinapism. Codeine is administered in moderate doses to relieve the cough and pain.

Pleurisy with Effusion (Subacute Pleurisy) and Empyema.

(*Purulent or Suppurative Pleurisy.*)

Frequency.—This form of pleurisy is common in infancy and childhood. The largest number of cases occur before the fifth year (Simmonds). The succeeding five years show the next greatest frequency. Israel found 29 per cent. of 206 cases to be purulent. Mackey estimates the purulent cases at 40 per cent. of the whole number in children, as against 5 per cent. in adults. Combining the statistics of Simmonds and Hofmokl of Vienna, this form is found to have greater frequency in the male sex. According to these authors, the left side is more often the seat of the disease. Simmonds found the disease to be bilateral in only 7 out of 175 cases. Of 170 of my own cases of empyema, 3 were bilateral. Of these the majority occurred before the fifth year, and 25 per cent. before the age of two years. The youngest patient was two months of age.

Etiology.—Primary pleurisy, whether suppurative or serous, is rare. The literature contains cases of acute effusion in the pleural cavity, in which there was apparently no exciting cause or primary lung affection. The etiology must in such cases remain in doubt. Infection may take place through so many avenues that it is difficult to point out the mode of entrance.

Pleuritis, serous or purulent, is generally secondary in infancy and childhood. All forms of lobar or bronchopneumonia may give rise to pleurisy, most of the cases being traceable to this source. The infectious diseases—measles, scarlet fever, pertussis, typhus and typhoid fever, diphtheria, forms of tonsillitis, retropharyngeal and mediastinal abscess, may precede or directly cause an attack of pleurisy. Chronic intestinal sepsis may cause empyema. In the latter case a pneumonia generally precedes the pleurisy or is present at the same time. In sepsis of the newly-born infant, there may be a complicating empyema. Osteomyelitis of the septic streptococcus variety may be complicated by purulent pleurisy. Tuberculous disease of the lung, actinomycosis of the lung, abscess of the liver, abscess in the mediastinum and abscess in the abdominal cavity involving the viscera, may cause pleurisy. Appendicitis may after the formation of abscess cause pleuritis by extension of the process along the coils of gut to the diaphragm. Finally, rheumatism may cause pleurisy of a serofibrinous nature. Exposure to cold and wet is undoubtedly a predisposing cause. In children, it is common to have a history of a fall or a blow occurring just prior to the attack of pleurisy.

Morbid Anatomy.—Pleurisies which accompany acute pneumonia are the most frequent. In these, there may be a slight injection of the pulmonary pleura and a loss of the normal lustre. Here and there a few fibrinous threads or adhesions may be found coursing over the surface of the pleura or running from the costal to the pulmonary pleura (dry or fibrinous pleurisy (pleuritis sicca)). In other cases, there is a thickened condition of both pleural reflections, caused by the deposit of fibrin on the surface. Sometimes the amount of fluid is small, while the pleura is very much thickened. The pleura itself may be little altered; underneath the fibrin, the lymph-spaces and blood-vessels may be dilated. In some cases there is also a serous or seropurulent exudate containing leucocytes, endothelial cells, and bacteria. The fluid may be clear or bloody, turbid or opaque, yellow or greenish, and thin or creamy in consistency. Large clots of fibrin may be found floating in the exudate. Adhesions may form pseudo-encapsulations of exudate, binding down the lung and preventing its expansion. In children, however, the tuberculous pleurisies are most likely to cause extensive thickening of the pleura. In addition to the deposit of fibrin on the costal and pulmonary pleura, there is a real inflammatory thickening of the tissue of the pleura itself, with a deposit of tubercle tissue. Serous or purulent exudate is encapsulated by adhesions, while the lung is bound down by layers of inflammatory tissue. In the tuberculous form the changes are progressive. In the acute inflammatory forms, the exudates are absorbed and the fibrinous deposit is organized into new connective tissue. In time the pleura may be restored to the normal. Adhesions, however, form an important factor in acute pleurisy of children. The pleura may in some cases be permanently thickened by a new layer of connective tissue persisting throughout life. There are forms of pleurisy not tuberculous in which this thickened condition not only remains, but extends from the pulmonary pleura into the lung along the interlobular tissue of the lung itself. There are induration and destruction of lung tissue. This induration is seen in connection with persistent bronchopneumonia. The amount of effusion (purulent) is sometimes quite large in children, and may reach 1000 to 5000 cubic centimetres (Simmonds, Hofmokle). In scurvy and morbus Werlhofii, blood may be effused into the pleural exudate.

Bacteriology.—Pleurisy or empyema is divided into several groups according to the class of bacteria found in the exudate. It is well established that the bacteria are the essential cause of the disease.

The first and largest group is that in which the pneumococcus of Fränkel, the lanceolate diplococcus, is found. These cases are called metapneumonic. They may occur during the progress of a pneumonia or after it has run its course. In some cases the process in the lung plays clinically a secondary rôle. The pneumococcus seems to occasion very little disturbance in the lung and to spend its force on the pleura. Thus within three days after the initial chill the pleura is filled with serous or seropurulent fluid. Netter found that of 28 pleurisies in infants and children 53 per cent. were due to the pneumococcus. In 212 cases of empyema I found the pneumococcus by culture in 75 per cent.

The second group comprises those cases in which the streptococcus alone, the stapylococcus, or the streptococcus with the pneumococcus or staphylococcus, is found. Netter found that 17 per cent. of his cases were of the streptococcus class; 10 per cent. of my cases were due to this micro-organism. In cases of the septic type, such as complicate sepsis of the newborn or osteomyelitis, or follow scarlet fever, the Streptococcus longus is found in the exudate.

These cases are severe. Six per cent. of my cases were caused by the staphylococcus. In 9 per cent. of my cases of empyema the streptococcus and pneumococcus were both found in the exudate. Although the pleurisies in which the streptococcus and staphylococcus are found may follow a pneumonia, they may also be secondary to a follicular amygdalitis, the exanthemata, typhoid fever, influenza, diphtheria, sepsis, and osteomyelitis.

FIG. 119.

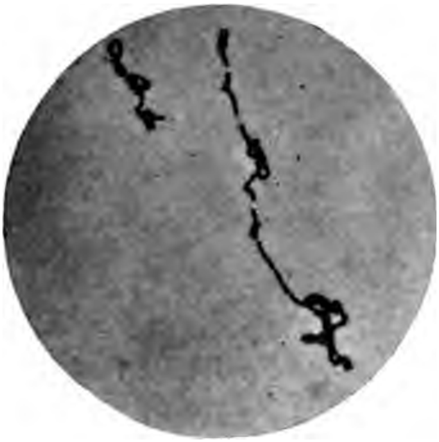


FIG. 120.

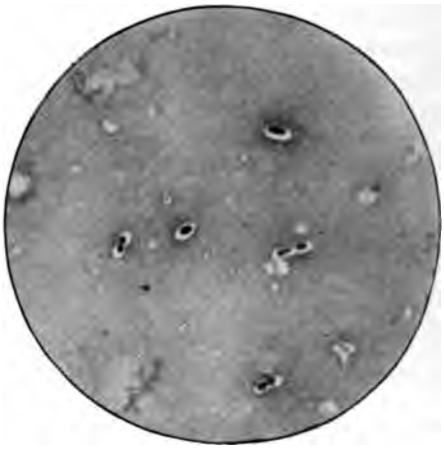


FIG. 121.

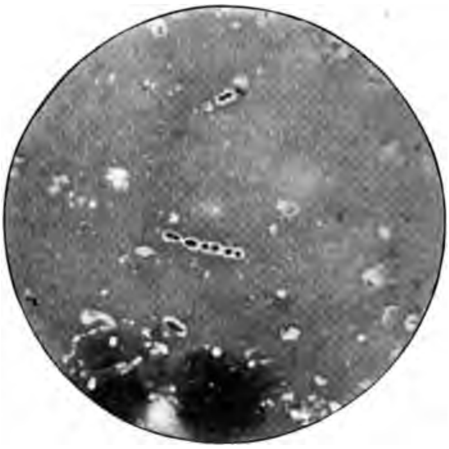


FIG. 122.

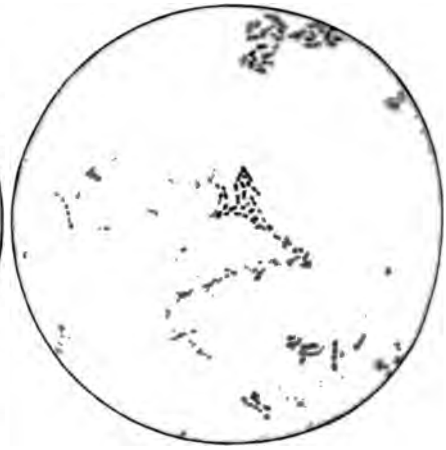


FIG. 119.—Streptococci from the pus of empyema: pure culture. Photomicrograph. × 1000.
FIGS. 120 and 121.—Pneumococci (Diplococcus lanceolatus) from the pus of empyema. Cover-glass preparations showing capsule. Photomicrograph. × 1000.
FIG. 122.—Pneumococci (Diplococcus lanceolatus); pure culture from the pus of empyema. Photomicrograph. × 550.

The third group of cases comprises those in which either the tubercle bacillus is found in the exudate, or the exudate is free from micro-organisms. The latter condition is frequently presumptive evidence of a tuberculous infection (Ehrlich). The tubercle bacillus was found in 1 per cent. of my cases, while in 3 cases the findings both by cover-glass spread and culture were negative. This would at most give a frequency of 2 per cent. for the tuberculous variety of pleurisy or empyema.

The last group is that in which micro-organisms other than those mentioned are found in the pleuritic exudate. Such cases have been observed in connection with typhoid fever in which the Eberth bacillus has been found. Escherich has found the coli bacillus in a case of empyema. I have seen one case of this kind. The bacillus of the saprophytic variety and that which causes a putrid empyema are found in cases of this fourth class.

The following table shows the relative frequency of the various forms of pleurisy and empyema with the varieties of bacteria in the exudate:

	Children.		Adults.
	NETTER 28 cases.	KOPLIK 212 cases.	
Pneumococcus	53.6 per cent.	75 per cent.	17 per cent.
Pneumococcus and Streptococcus	3.6 "	9 "	2.5 "
Streptococcus	17.6 "	10 "	53 "
Staphylococcus		6 "	1.2 "
Putrid	10.7 "		
Tubercule bacillus	14.3 "	2–3 "	25 "

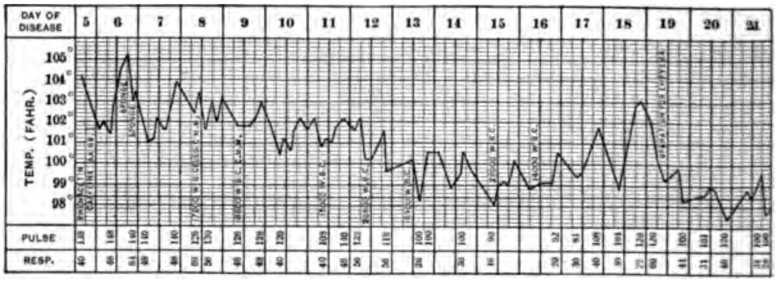
The most important fact to be deduced from the statistics is that while tuberculous pleurisy in children has a frequency of 2 to 3 per cent., adults show a much greater frequency, many of the streptococcus cases being tuberculous in the latter subjects. This figure added to the number of cases in which tubercle bacilli are found in the exudate would bring the frequency in the adult to at least the 45 per cent. given by Bowditch as the relative figure.

The **physical characteristics** of an effusion in the chest are of clinical importance. An effusion if purulent has usually the gross physical characteristics of ordinary pus. In some cases the effusion is at first clear and serous, but is subsequently seen to be purulent without the occurrence of any extraneous infection. In other cases the effusion may be a cloudy serum, which on exploratory puncture is after a few days found to be purulent. In rare cases the effusion or exudate in the pleura is hemorrhagic. An effusion of that character has not the same significance in children as in adults. In the latter such effusions may be tuberculous or due to some morbid growth of the pleura; this is not necessarily the case in children. I have had a number of cases of hemorrhagic effusion into the pleural cavity. In none of them was there a tuberculous element.

In all, streptococci were found in the effusion, and in some the admixture of blood could be traced to a scorbutic tendency. In one case, in an adolescent with localized effusion of a hemorrhagic nature, there was an actinomycosis of the pleura and lung. The history of this case was not that of an effusion of an acute, but of a subacute chronic nature.

Symptoms.—There are no symptoms characteristic or pathognomic of effusion in the pleura or empyema. The condition is in most cases masked by the symptoms of the causal affection. Cases following a pneumonia set in with a chill or a rapid rise of temperature, with which there may be a convulsion followed by stupor or cerebral symptoms. After this onset the fever continues, ranging from 103° to 105° F. (39.4° to 40.5° C.), the pulse being 140 to 180. There will be cough, great dyspnœa, and pain in the chest, which is especially manifest when the infant or child coughs. The breathing is shallow. After a few days the acute symptoms subside, the fever becoming remittent. The temperature may be nearly normal. The dyspnœa continues, although the temperature and pulse may be normal during part of the day.

FIG. 123.



Lobar pneumonia; fall of temperature, by lysis; gradual rise after the thirteenth day, due to empyema. Operation on the nineteenth day. Recovery. Boy, four years of age.

In some of the cases the effusion becomes apparent on the eighth day; in others a purulent effusion is found in the chest on the twelfth or fourteenth day of the disease. The effusion, which finally becomes apparent in the chest, has been coincident in its onset with a pneumonia—there has been a pleuropneumonia. The process in the lung, however, takes a secondary place in the clinical picture when the effusion in the pleural cavity has accumulated.

There is another set of cases in which the course of the disease is insidious. The patient may at the onset have had for two or three days a febrile movement which has subsided, leaving the child not quite well and with a slight febrile movement toward evening, a slight hacking cough, and some little pain in the chest on exertion. Langour and loss of strength are progressive. There may be exhausting sweats at night. Examination of the chest will reveal an effusion.

The metapneumonic pleurisies in infants and children have a characteristic course. The patient has a typical pneumonia. The temperature on the ninth, tenth, or thirteenth day may drop to the normal or subnormal, the respirations continuing high. A gradual rise of temperature follows, with physical signs of fluid in the chest (see Fig. 123). The pulse and respirations rise with the temperature. Toward evening there may at times be chilly sensations. Exploration may discover fully developed effusion in the chest, serous or purulent according to the severity of the pleuritic infection. As a rule the younger the subject, the more likely is the effusion to be of a purulent nature. The duration of the effusion in the chest will also be a guide in determining its nature. An effusion occurring after pneumonia in a young infant and persisting for a week after the pneumonia has run its course, is likely to be purulent.

Diagnosis.—There are some symptoms, such as continued dyspnœa, a slight or troublesome cough, exhausting sweats, and a distinctly intermittent range of temperature, which in all cases of pleurisy should direct attention to the chest. None of these symptoms is, however, pathognomonic of pleurisy, serous or purulent, since they may be found in other pulmonary conditions. The diagnosis of pleurisy with effusion or empyema should take into consideration not only the rational symptoms, but also the physical signs.

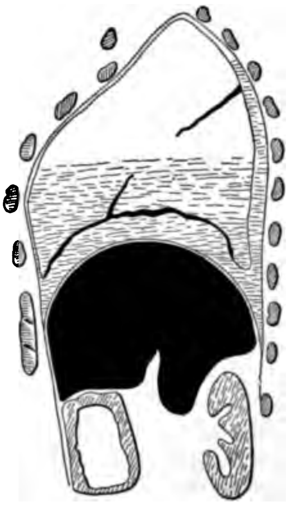
The **physical signs** of pleurisy with effusion and of empyema are identical.

FLUID IN THE CHEST.—*A.* The chest partly filled with fluid. *B.* The chest full of fluid.

A. The Chest Partly Filled with Fluid.—It is assumed that the greater part of the fluid is in the lower portion of the chest (Fig. 124). In children and infants it does not cause displacement of the viscera.

Inspection may show fulness of the lower part of the affected side; the lower part of the chest moves less than the normal side.

FIG. 124.



Pleural cavity partly filled with fluid.

Palpation.—Vocal fremitus will be felt over the upper portion of the chest in front or behind, and will be lost over the lower portion.

Percussion of the chest in front will often give an exaggerated hyperresonant tone over the upper lobe of the lung. Behind, there is almost always dulness to a greater or less degree above over the scapula, due either to thickening of the pleura or to an exceedingly thin layer of fluid. This dulness can be distinguished from dulness due to other causes by firm percussion which will elicit the pulmonary note of the underlying lung. Below, over the fluid, the dulness changes to complete flatness.

Auscultation.—The voice and breathing may be heard over the whole side with as much intensity as on the healthy side, or with diminished intensity below the level of the fluid. Râles, generally pleuritic crepitations, may be heard above the level of the fluid. Bronchial breathing and voice may be heard over the fluid or at the level of the fluid, but this sign is not absolute.

Diagnosis to justify needle exploration must be based on absence of vocal fremitus over the fluid and its presence above the fluid, dulness behind above the fluid, which on firm percussion gives a faint pulmonary tone and flatness over the fluid with slightly increased resistance to the percussing finger.

Note.—The method of examining infants for fluid is invariably that indicated in the earlier part of the book. It is a mistake to examine the infant as it lies in the lap of the mother, for in this position the fluid will gravitate behind. When the infant lies on the face, the fluid will again gravitate to the anterior part of the chest and thus not be made out. In the earlier stages of pleurisy the fluid only partly fills the thorax. On account of the small size of the thorax in infants, it is impossible to determine the change of level of the fluid by changing the position of the patient.

The resonant note over the lung apex in front should, in the presence of dulness behind and flatness below, always arouse suspicion of fluid, for in these cases the lung seems to be compressed upward, forward, and inward, thus causing the vesiculotympanitic note in front and above.

The chest is partly filled with fluid, as is shown in Figs. 125 and 126. I have quite frequently found this condition in infants and children who have for a long time lain on the back, and in whom adhesions seem to have kept a layer of fluid in the position shown in the figure. It will be assumed for illustration that the right side is affected:

On *inspection*, fulness of the intercostal spaces on that side may be detected; the movement of the thorax is labored, and the intercostal spaces may be drawn inward on inspiration.

Palpation.—Vocal fremitus due to the lung's being in contact with the chest-wall may be present over the anterior aspect of the chest. Posteriorly, the fremitus will be entirely absent.

Percussion.—Anteriorly, the note may be vesiculotympanitic; posteriorly, there is complete dulness over the whole chest, which is more marked below. There is rarely the flatness obtained when the chest is full or half full of fluid. There is also resistance to the percussing finger.

By percussing firmly the note of the lung beneath will invariably be elicited; breathing-sounds and voice-sounds will be heard as normal or distant.

Pleuritic crepitations may be heard over the whole affected side; there is no displacement of the liver or heart on the left side.

Diagnosis of fluid before exploratory puncture must rest on the complete or partial absence of fremitus behind, and complete dulness or flatness. The quantity of fluid is small; there is less resistance to percussion than when it is large.

FIG. 125.

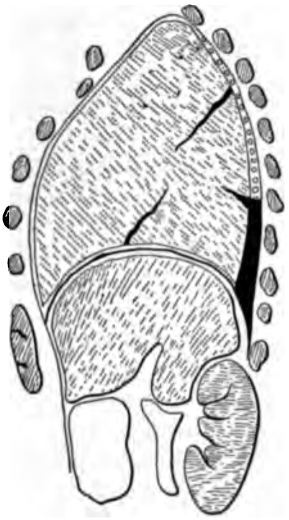
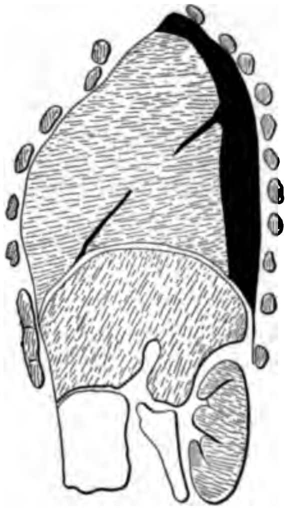


FIG. 126.



Fluid in a thin layer posteriorly in the pleura.

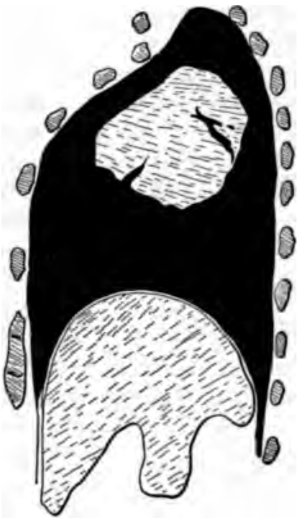
(*B.*) *The Chest Full of Fluid* (*Right Side*).—On *inspection*, the objective signs of intense or moderate dyspnœa are found: The chest on the affected side is immobile; the intercostal spaces are retracted with each inspiration; the affected side bulges visibly.

Palpation.—Vocal fremitus is lost over the whole side in front and behind. In rare cases a little fremitus is felt.

Percussion.—Ordinary and firm percussion give a flat note over the whole chest in front and behind; the resistance to the percussing finger is wooden. In front, flatness may be present over the apex of the lung (Fig. 127). In some cases in children a sound over the apex of the lung, which resembles the cracked-pot sound over cavities in adults, may be obtained. It may be due to lung compression. In other cases the resonance in front, over the lung of the affected side is vesiculotympanitic, owing to the pushing upward and forward of the lung and to its distention.

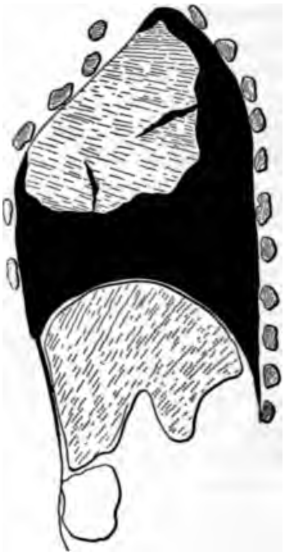
Displacement of the Pleural Fold underneath the Sternum.—A very important aid in the diagnosis of fluid in either side of the chest is the displacement of the line of the reflection of the pleura in front. Normally the pleuræ of both sides meet underneath the sternum in the median line. Above, at about the level of the second rib, they depart gradually from each other. If there is a large amount of fluid in the right chest, the pleural fold of that side becomes distended and displaced to the left, and may be marked out above the heart by dulness to the left of the midsternum. If the left chest is full of fluid, the left pleural fold is displaced to the right and there is distinct dulness or flatness above, to the right of the midsternum (Fig. 129).

FIG. 127.



Pleural cavity full of fluid. Flatness anteriorly and posteriorly.

FIG. 128.

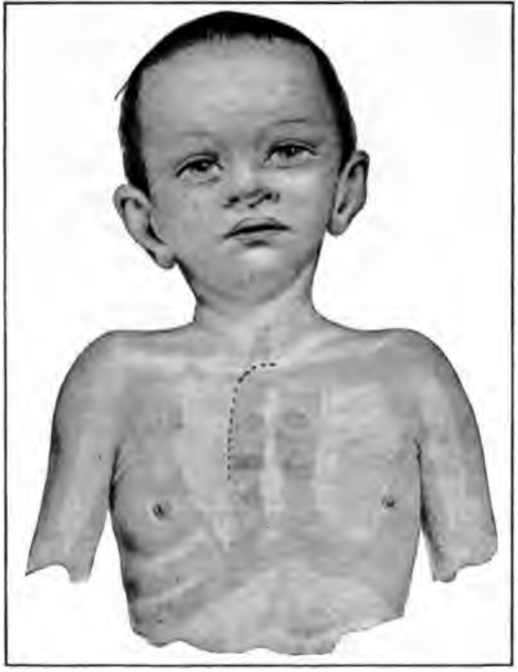


Pleural cavity filled with fluid. Lung displaced upward and forward. Resonance anteriorly over the apex, either vesiculo-tympanitic or of the cracked-pot quality.

Auscultation.—Auscultatory signs in infants and children are most puzzling when the chest is full of fluid, and little diagnostic value can be attached to them in some cases. The chest may be full of fluid while the breathing and the voice may be heard as on the unaffected side, and pleuritic crepitant râles or crepitations may be heard over the whole chest behind. In other cases, the breathing may be indistinct and distant, and in the lower part of the chest lost entirely. The voice may be bronchophonic in certain localities; it may be of this quality over the whole diseased side of the chest behind, or the tubular sound may be conducted to the healthy side. The voice may be normal above and heard faintly below, toward the base of the lung.

Diagnosis before exploratory puncture rests mainly on (*a*) complete absence of fremitus; (*b*) absolute flatness on percussion with resistance to percussion; (*c*) bronchial voice and breathing over the whole chest behind; (*d*) hyperresonance over the apex, and displacement of viscera, and of the pleural fold in front.

FIG. 129.



Displacement of the left pleural fold in effusion (empyema) into the left pleural cavity; flatness to the right of the midsternum as indicated.

DISPLACEMENT OF VISCERA.—*Liver.*—In infants and young children the presence of fluid may be indicated by displacement of the liver downward on the right side. I have been able to verify the displacement in cases in which large amounts of fluid were present. In infants, the liver is so large and the projection below the border of the ribs so undetermined, that it is difficult to estimate the exact amount of displacement. The chest is so easily dilated that an ordinary amount of fluid accommodates itself without markedly displacing so heavy an organ as the liver. In children I have been able to make out a displacement of the liver downward before the evacuation of large quantities of fluid. Displacement is of confirmatory value in diagnosis.

Heart.—The heart-apex may be displaced toward the median line by fluid in the left pleural cavity. In older children also when the amount of fluid is large the apex is displaced and lies beneath the lower part of the sternum. A small amount of fluid will not always cause displacement, but will find its way around the heart.

Remarks upon the Diagnosis of Fluid in the Chest, with Exceptional Signs.—It is not always easy, even for the expert, to decide without puncture as to the presence or absence of fluid in the chest of infants and young children. The following signs will be of service at the bedside.

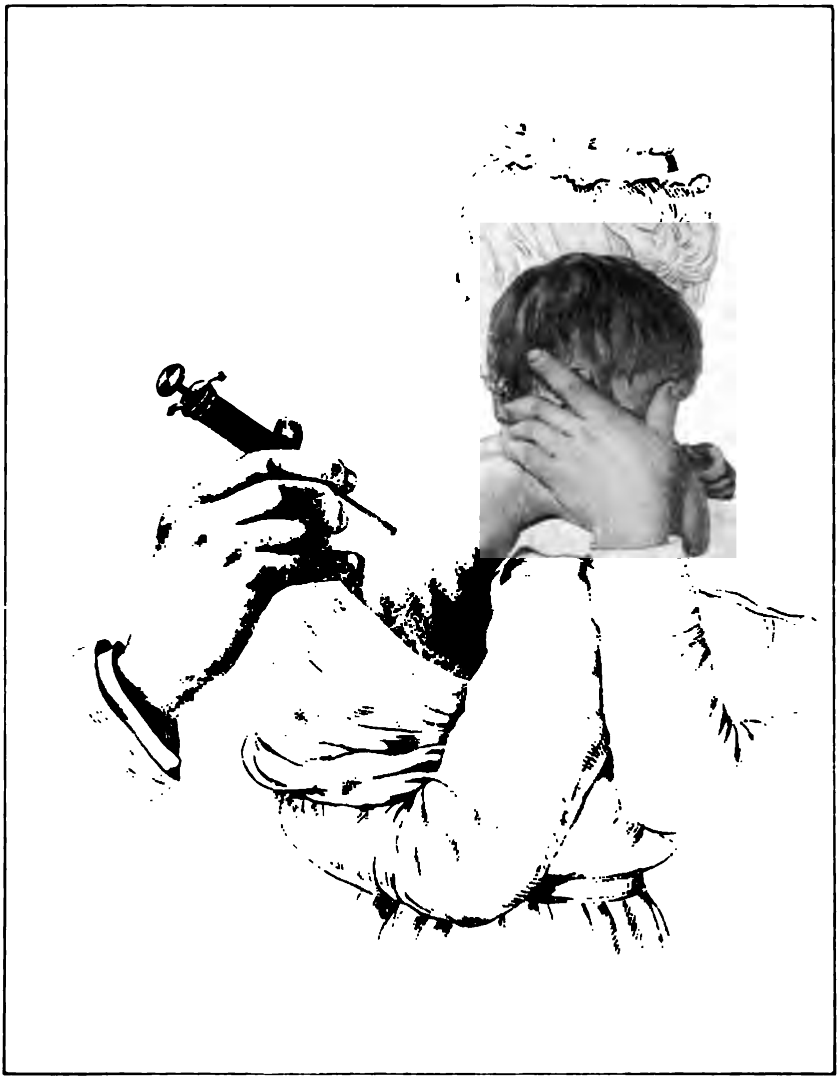
DURATION OF ILLNESS.—If an infant or child has been ill for two weeks or more with signs of pneumonia during the early part of the disease, the physician should be watchful in the presence of the following conditions: If the temperature does not fall, but though remitting still continues; if the signs of consolidation of a small or large area give place to dulness or flatness over a whole side behind, with *bronchophony* over the whole side—for if the condition of the infant is tolerably good, it is evident that such bronchophony may not be due to the total consolidation of the whole lung; if there is displacement of viscera, chiefly of the liver or the heart; if there is drawing inward of the intercostal spaces during inspiration, with real immobility and bulging of a side and dulness or flatness and loss of fremitus.

Fluid is very rarely encapsulated in a small area behind, about the midregion of the chest. Such areas are usually areas of persistent bronchopneumonia. In most cases, there is localized dulness, above and below which there is vesiculotympanic resonance, normal pulmonary resonance or exaggerated resonance. There is distinct respiratory movement of the affected side. On the other hand, a collection of fluid between the lobes of the lungs (interlobar) may give a localized flatness and all the auscultatory signs, such as bronchial voice and breathing, of a local collection of fluid.

There are certain localities in which the diagnosis of fluid must be made with reserve:

a. In a case on which I operated, fluid was found posteriorly over the situation of the upper lobe of the right lung. The fluid was completely shut off from the rest of the pleural cavity by a

PLATE XVIII.



Showing the correct position of the child and operator in making an exploratory puncture for fluid in the pleural cavity. The plate is not intended to illustrate the point of puncture, which is always at the discretion of the operator.

membrane stretching from the thoracic wall to the interlobar fissure of the lung. Post-mortem showed the case to be tuberculous, the lung on the affected side being the seat of persistent tuberculous bronchopneumonia. I have seen similar cases which were metapneumonic.

b. Fluid over the upper lobe in front only, is rare. I have seen one case, but no operation or verification was permitted.

c. Fluid over the lower lobe of the lung, in front on the right or left side without corresponding signs behind, is uncommon.

d. Circumscribed collections of fluid behind over the middle region of the lung or toward or in the axillary line are exceedingly uncommon.

e. In the chapter on the physical signs of pericarditis, it will be shown how a pleurisy or empyema on the left side may be mistaken for pericarditic effusion.

Physical signs having led the physician to suspect fluid, the chest should be explored for two distinct reasons : to determine absolutely the presence of fluid, and to ascertain whether it is serous or purulent.

Diagnostic Exploratory Puncture of the Chest.—The INSTRUMENTS necessary are an exploring needle, a millimetre in calibre, and a large barreled aspirating syringe. The needle should not be too short, else it may snap off in the chest. The needle and syringe are boiled for a few moments before being used. The patient is held in the arms of the nurse or mother, so that the posterior aspect of the chest may be exposed. Older children may sit on a table. The chest is scrubbed with soap and water, washed off with ether, then with alcohol, and finally with a solution of sublimate (1 : 2000). The arms of the infant or child are firmly held and the chest steadied in such a manner that should the patient move suddenly the needle will not break in the chest (Plate XVIII.).

INTRODUCTION OF THE NEEDLE.—The chest is again percussed and the needle introduced into the intercostal space in which percussion elicits the most marked dulness or flatness. This rule should be invariably followed; the needle should not be introduced into any particular intercostal space. On the right side the physician should avoid putting in the needle too low down (liver); on the left side he should avoid introducing it too deeply for fear of wounding a large vessel at the root of the lung. The needle should not be entered too near the vertebral column. The needle having been introduced one or two centimetres, the piston is drawn and held thus a few seconds. Sometimes the fluid is thick and does not flow freely into the syringe. The syringe should not be introduced and then withdrawn and pointed up and down in various directions in quest of fluid, for fear that the struggles of the patient, even if he is firmly held, will cause puncture of the lung and bloodvessels. The needle should be withdrawn as rapidly as it was introduced and the whole operation completed in less than a minute. The external wound is covered with a small strip of iodoform gauze held in place with rubber plaster. The needle while in the chest should be held loosely. If it is held firmly, any sudden movement of the patient will cause it to break off in the chest. The needle should not be introduced too deeply for fear that it may enter a dilated bronchus and withdraw purulent secretion which may be mistaken for empyema, or that it may wound the lung and cause hemorrhage or pneumothorax.

Perforating Empyema.

An empyema may perforate externally. In that case there will be an extensive infiltration of the tissues external to the ribs on the affected side, resembling a large phlegmon, and the signs of fluid will persist. If the perforation occurs on the left side, the movements of the heart are likely to be conducted to the external swelling, and there is then what has been called pulsating empyema. The empyema may perforate through the lung, and the signs will then vary with the length of time during which the perforation has existed. It is customary for writers to repeat one another in recounting the physical signs of pneumothorax in a chest in which fluid (pleurisy or empyema) is present. In infants or very young children the following classical signs of pyopneumothorax observed in adults are not commonly found: amphoric breathing, amphoric voice, metallic tinkle, and succussion-sounds. My cases were in children under two years of age. The perforation in the lung must have been too small or too valvular to permit of the entrance of much air into the pleural cavity. These cases at first showed all the signs of the condition which was proved, on introducing the needle, to be empyema. Operation being refused, after a few weeks (three months after the beginning of the disease), the signs changed as follows:

Periodic expectoration of large quantities of pus following coughing spells.

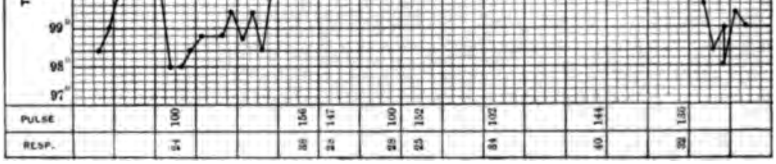
Fremitus diminished over the whole right side and almost lost below.

Dulness over the whole side in front and behind, with tympanitic note on deep percussion only. Voice normal; breathing normal—at least not varying from that on the healthy side. In the intervals of expectoration, there were in some cases bronchial voice and breathing.

No succussion-sounds, no tinkling, no amphoric signs. The classical signs seen in adults are met in children above five years of age.

Course and Termination.—Pleurisy with effusion and empyema have been considered together, because, in infants and children under two years of age, the effusion in the chest may at first be serous, but subsequently change into purulent exudate. A serous effusion may be followed by a purulent one; it may remain serous and be absorbed as such. Thus it is best, especially in infants, to introduce an exploring-needle into the chest to determine the nature of the fluid as soon as its presence is suspected. In older children also, this may be done at the outset. If a clear fluid is at first obtained and the symptoms do not retrograde within a short time, the needle should be again introduced to determine whether the fluid has remained serous. It is frequently found to be purulent although no infection has occurred as a result of the first puncture. With ordinary cleanliness, the possibility of infecting a serous effusion in the chest and thereby causing it to become purulent is very slight. Purulent effusion appearing after the first exploratory puncture has shown the effusion to be serous, may be due to two causes: either to continuance of the pleuritic inflammation, or to the fact that if the infant or child has lain quietly in bed the purulent elements of the effusion have gravitated to the lower portion of the chest, leaving a clear serum above at the level of the puncture.

FIG. 130.



Empyema, left pleura, followed thirteen days after operation by bronchopneumonia at the apex of the right lung. Male child, twenty months of age. Recovery.

The **prognosis** of pleurisy with effusion and of empyema in infants and children is good. If treated in the proper manner, it is not more serious than the original causal affection. In private practice, the patient being under constant supervision of the physician, the outlook is very good. An effusion can be discovered early and the patient relieved. In hospital practice the results are still good if the cases are simple and come under treatment before systemic infection has taken place. In my service of 120 cases of all kinds, there were 20 deaths, 4 of which occurred from one to five days after operation. Sepsis had been present before operation and caused the fatal issue. The septic cases therefore give an unfavorable prognosis, as do also those of a tuberculous nature. In the latter, as in other forms of tuberculosis in children, the outlook is better than in the adult and recoveries are not infrequent.

Of the 20 cases of death after operation for empyema, bronchopneumonia either persistent or recurrent caused the fatal issue in 11, general sepsis in 2, marasmus and ulcer of the duodenum in 1, and cerebral embolism in 2. A complicating pericarditis of a suppurative nature may cause death. It is not always possible to diagnose this condition during life. The complication most to be feared in empyema is a bronchopneumonia involving either lung. In many cases the bronchopneumonia is present at the time of operation, or it may come on a week or two afterward during apparent convalescence.

The prognosis of tuberculous empyema is not so unfavorable in children as in the adult. In the former, empyema of a tuberculous nature, like other forms of tuberculosis, may with skilful management make an apparent recovery, though with marked deformities of the chest-wall. In this form of empyema the pleura is thickened, binding down the lung and thus preventing expansion. Extensive rib resections thus become necessary in order to close up the suppurating cavity left by the unexpanded lung.

Treatment.—If on exploratory puncture a serous exudate which only partly fills the pleural cavity is found, the expectant plan is followed. The bowels are kept open with an enema or a saline cathartic is administered daily. For this purpose a saline enema, or in older children a teaspoonful of Carlsbad salts in warm water mixed with milk, is efficient. Local vesication is not needed nor is it advisable. The effusion is absorbed if the patients are kept quiet and the diet is easily assimilable. Citrate of potassium in grain v (0.3) doses every three hours may be given to older children. If the fluid increases in quantity, fills up the chest, causes dyspnœa or pressure symptoms, and is serous in character, the chest should be aspirated. The best form of aspirator for the practitioner is the Potain. The patient is aspirated in the sitting posture. The chest-wall having been cleansed, the needle is introduced in the posterior axillary line toward the lower third of the chest cavity. It is not withdrawn until the flow has ceased or the lung can be felt against the needle in the pleural cavity. As soon as this occurs the needle is withdrawn and the puncture opening covered with a piece of iodoformized gauze. It sometimes happens that there are signs that the chest is filled with fluid and yet very little flows into the instrument. In such cases the needle should be withdrawn and introduced into the chest-wall at another point. The coughing attack which occurs during aspiration will subside on the patient's taking the recumbent posture. If the chest is quite full of fluid, it is well not to empty it entirely. Sometimes alarming syncope with other signs of cardiac weakness, such as cyanosis, has supervened. If a limited quantity of fluid is removed, the absorption of the rest will follow rapidly.

A daily saline cathartic is given; the patient is kept quiet and allowed a nutritious and easily assimilable diet. The administration of salicylate of sodium may hasten absorption, especially in cases in which there is a rheumatic history. If there is pain or a harassing cough, small doses of codeine should be given.

Empyema.—When the presence of pus in the chest is once established, it is imperative that it be evacuated with the least possible delay. In infants and children it is not advisable to temporize by first performing aspiration. Retention of even a limited quantity of purulent exudate in the pleural cavity not only leads to emaciation and physical weakness as a result of continued fever, but general sepsis may also result. Aspiration is not efficient, and is to-day practically abandoned as a mode of treatment. The physician may either incise the intercostal space or resect a rib to obtain drainage.

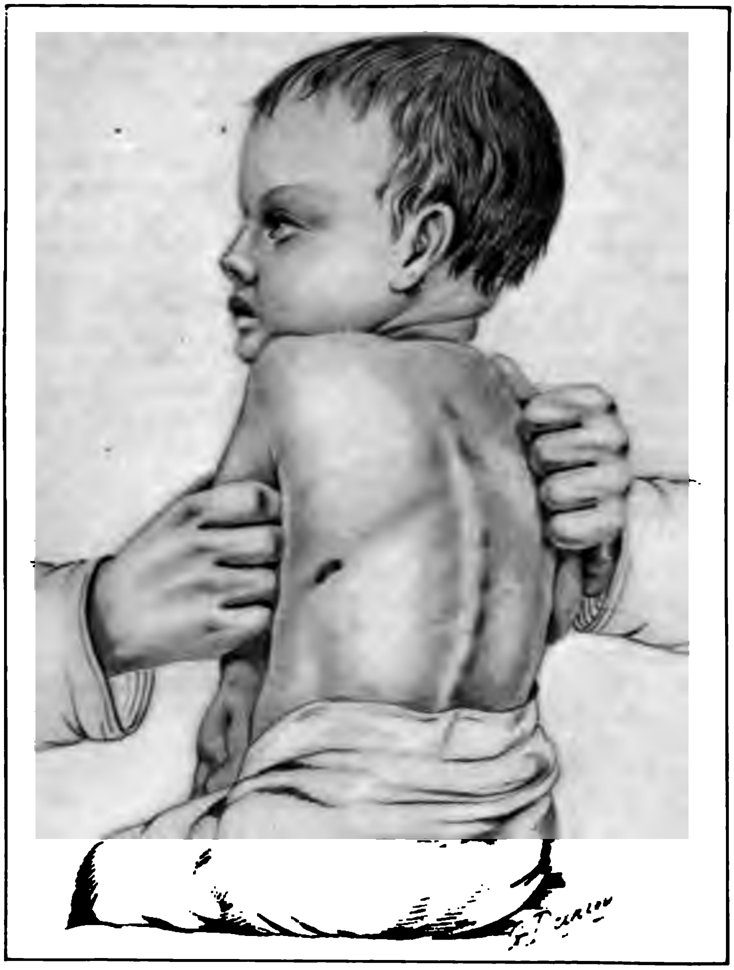
Simple incision in the intercostal space is efficient in many cases of empyema occurring in the first eighteen months of life. In these frail patients, excision of the rib has been sometimes accompanied by discouraging results.

The greatest number of deaths after any operative procedure for the relief of empyema occur in children under the age of eighteen months. The strength of the patient should be supported as much as possible. A general anæsthetic is not necessary for patients under this age. I find that bronchitis and pneumonia very frequently result from the general use of anæsthetics in young patients. Local anæsthesia is all that is needed. Ethyl chloride in tubes is very efficient. The surface of the chest is carefully cleansed with soap and water, alcohol, ether, and sublimate. An incision two inches long or thereabouts is made obliquely in the tissues over the intercostal space. The space in which a needle has been previously introduced and pus found is chosen. The exploring-needle is always introduced just before operation. Frequently, although pus has been withdrawn from the chest, at a second aspiration none can be found. The theory is that either there was a small localized collection of pus at the first point of aspiration, or that the needle entered a bronchus and withdrew secretion collected there.

On the right side the incision should not be too low, else a tube cannot be retained in the chest on account of the high position of the diaphragm. The seventh or the eighth space in the posterior

axillary line is the best location if pus is present at this point (Fig. 131). On the left side, incisions should not be made too far forward, else the drainage-tube may impinge against the pericardium.

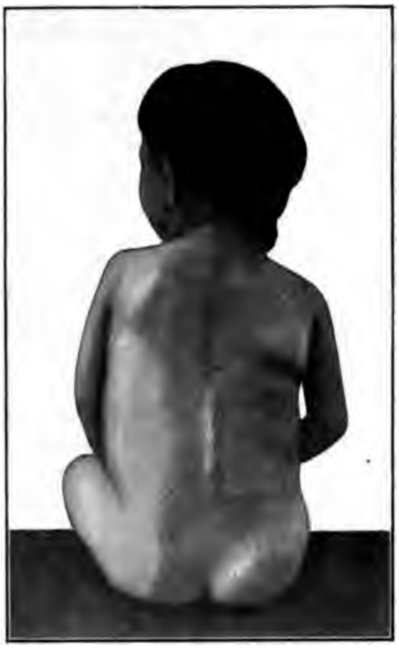
FIG. 131.



Empyema, site of incision in line with the angle of the scapula.

The superficial tissues having been incised, the intercostal muscle is incised, the operator keeping as nearly as possible in the median line of the intercostal space and avoiding the lower border of the upper rib, yet not cutting too close to the lower rib. When the vicinity of the costal pleura is reached, a closed dressing-forceps is introduced into the pleural cavity and opened to widen the puncture. A small drainage-tube or two small tubes are placed in the pleural cavity and prevented from falling into the pleural space by safety-pins passed through them at the distal ends. The pus is not evacuated at the time of operation. The sudden evacuation of fluid which has been retained in the chest for a long time is apt to cause untoward syncopal symptoms. Gibson has made the excellent suggestion that as soon as the pleura is opened the drainage-tube should be quickly introduced into the chest, the gauze dressings applied, and the pus allowed to escape gradually into the dressings. The dressings consist of a pad of gauze around the tubes, covered by a dry sterilized gauze dressing which is renewed every day. The chest should not be irrigated. No instrument should be introduced into the chest cavity to loosen adhesions. The whole operation is extremely simple, and should not occupy more than a few minutes. Children under five years, and even older ones may be treated by this method. In the older subjects, however, the chest-wall is not so resilient; there are adhesions, and if they are numerous and clots are abundant in the exudate a subsequent excision of the rib may be

FIG. 132.



Exsection of rib for empyema on the right side. Shows the resulting deformity. Five weeks after operation. Child, four years of age.

necessary. On the other hand, the main object of the practitioner in these cases is to evacuate the pus, and incision will accomplish this quite as well as the other operation. If subsequently more drainage is needed, the patient will be stronger and better able to stand the more serious procedure.

Incision is therefore the practitioner's operation even in older children, with whom anæsthesia must, however, be used. Chloroform is the safest and most easily taken; very little need be used. As soon as the skin incision has been made, anæsthesia should be suspended.

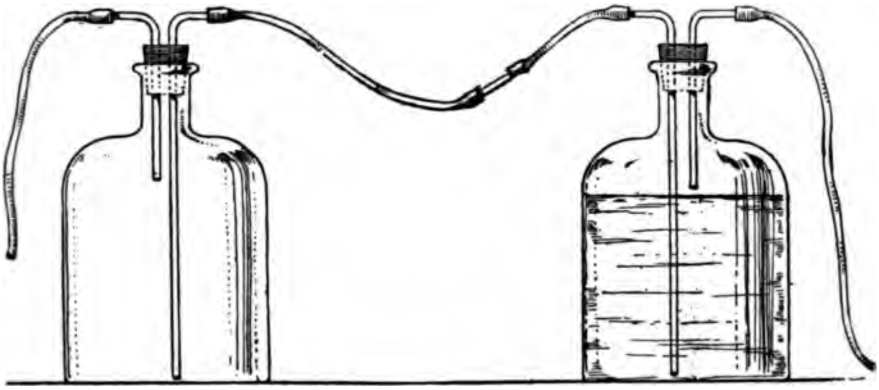
I perform excision of the rib in the cases of children above the age of eighteen months, unless there is a contraindication. Severe pneumonia, high fever, cardiac weakness, acute pericarditis or endocarditis, as complications, are contraindications. In such cases incision alone is performed. I excise the rib in the usual way, taking two or three centimetres of rib subperiosteally and incising in the midline of the posterior layer of periosteum to enter the pleural cavity. The finger is not inserted into the pleura to loosen adhesions. After the pleura is opened, double drainage-tubes are introduced by Gibson's method, as in the operation of simple incision.

Sinus.—After incision or resection of the rib, a suppurating sinus may remain for months. If a probe introduced into a sinus of this kind impinges against callus or denuded bone, a so-called secondary operation is necessary to take out the denuded rib or callus. This involves a difficult surgical procedure, which it is not necessary to describe here. A sinus of this form will not close until the bone is removed. Temporizing only subjects the patient to the dangers of prolonged suppuration (amyloid degeneration).

Adhesions binding down the Lung.—There is another class of cases in which a large amount of fibrin has been thrown out on the visceral pulmonary pleura. The lung is thus cramped by an envelope of thickened pleura and cannot expand. A large suppurating cavity or a suppurating sinus is left between the pulmonary and costal pleura. This cavity must be made to close. In such cases the patients are allowed to be up and about. They are taught to blow colored fluids from one bottle to another in the way described by James, of New York (Fig. 133). Two bottles of equal size, each half filled with the fluid, are used. In simple cases this method is very efficient; in others it is of no avail. The operation of taking out two or more ribs with the intervening pleura must then be performed. In other cases a more extensive operation—the so-called Estlander, in which large pieces of several ribs are excised with the intervening costal pleura—is necessary. If the lung is firmly bound down by a coating of fibrin, the chest-wall must be opened by reflecting a flap of several ribs and the soft parts. The pleura is peeled off the lung according to the method of Delorme. The lung expands, the costal flap is sewn back in its place, and the chest sinus is in time closed as a natural consequence.

The question of irrigating the pleural cavity in the treatment of empyema after operation has been much discussed. As a rule, if the temperature drops after operation and remains low, and the discharge is not fetid, no irrigation is indicated. If, however, there are rises of temperature after operation, with a profuse or fetid discharge, the chest should be irrigated once daily with normal salt solution.

FIG. 133.



James' apparatus for expanding the lungs in empyema.

Bilateral Empyema.—The treatment of bilateral empyema will tax the judgment of the physician. One side, preferably the left in order to relieve the heart, is first operated on by incision or rib exsection; the other side is aspirated, and again aspirated if the fluid or pus accumulates. After a week adhesions will have formed on the operated side, and the strength of the patient will warrant interference on the opposite side. When this is accomplished, the opening on the operated side must be closed by some device, such as a pad of gauze on which is placed rubber tissue covering, and the second side may be operated on by rib exsection or incision.

I have followed this method in two cases without serious accident. The interval of a few days between the operations is sufficient to allow adhesions to form on the operated side to such an extent that, when the second side is opened, the lung of the side first operated on does not collapse. If the sides are operated on simultaneously, the consequent partial collapse of both lungs causes marked symptoms of asphyxia.

Hemorrhagic Pleurisy.

Simple hemorrhagic pleurisy is not uncommon. It is seen in pleurisy following simple pneumonia, influenza, the exanthemata, and in infants or children in whom there is a tendency to scorbutus. Cases which appear to be rheumatic have been published (Starck). The hemorrhagic form of pleurisy with effusion may occur in very young infants (Lewin, eleven months) or in young children. I have met a number of cases in children who subsequently made a complete recovery, and in whom I could find no tuberculous tendencies. The prognosis in this form of pleurisy is therefore much better in children than in adults. In the latter, a hemorrhagic pleurisy is frequently indicative of a tuberculous factor in the etiology.

Hemorrhagic Empyema.

Hemorrhagic empyema is also not uncommon in infants and children. During the past year I have met four cases in which there was a hemorrhagic exudate. In one case the child was pale, though not emaciated. There may have been a scorbutic element. In another case, in a boy, no such etiology was indicated. In a third case, in a girl, the child was much reduced in health. In three cases the hemorrhagic discharge persisted for days after the chest was opened and streptococci were found in the exudate. In one case the discharging pus was for weeks tinged with blood. In none of the cases were tubercle bacilli found in the pleuritic exudate. Three of the cases made a very good recovery. In these cases also I am inclined to believe that tuberculosis is not always an etiological factor.

Subphrenic Abscess or Pyopneumothorax Subphrenicus.

The positive diagnosis of subphrenic abscess should be made with reserve, because no pathognomonic symptom or physical sign of the disease is known. It is a very valuable fact that in 50 per cent. of the cases thus far recorded, the abscesses have contained gas or air. The condition is rare (Maydl) in adults and more so in infants and children. The abscess is situated beneath the diaphragm, and between that organ and the liver. It pushes the diaphragm upward, and may thus encroach on the pleural space and simulate a real pyopneumothorax. An area in the lower part of the thorax, which may give tympanitic resonance or tympanitic dulness from the second, third, or fourth rib downward, is thus caused. This resonance may even include the liver, which is displaced downward. Over the region of tympanitic resonance, especially posteriorly, the normal vesicular breathing is absent on expiration and present over the area on deep inspiration. It is a peculiarity of the condition that there may be amphoric breathing and metallic tinkle over the area, while anteriorly, just above it, from the second to the fourth rib, there is a sharp transition and normal breathing is heard. Behind, however, on deep inspiration, even over the region of tympanitic resonance, normal breathing may be heard over the lower part of the chest. Over the situation of the abscess the metallic tinkle and succussion-sounds may also be heard. As has been stated, the liver may be displaced downward, crepitations are heard anteriorly over the liver (perihepatitis), or it may be impossible on account of intestinal conditions to make out the lower border of the liver. I have seen a subphrenic abscess on the left side displace the left lobe of the liver and the spleen downward. The heart is not displaced inward if the abscess is on the left side, but if displaced at all, is so in an upward direction. The lower thorax region may show no abnormalities to inspection, while the upper abdominal region may be normal, painful to pressure, or slightly œdematous.

Diagnosis and Treatment.—Exploratory puncture is resorted to in all of these cases. Diagnosis will be aided if the fluid obtained contains, in addition to pus, elements which denote the origin of the abscess, such as food particles, feces, histological débris or pigment from the liver. In many cases the liver suffers from the vicinity of the abscess.

The treatment is surgical.

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SECTION VII.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

Pericarditis.

PERICARDITIS is an inflammation of the pericardium due to infection, which may take place through the blood- or lymph-channels or may occur through contiguity to infected areas in neighboring structures. The existence of primary pericarditis or so-called idiopathic pericarditis apart from rheumatism or infection is a matter of doubt. It is therefore to be regarded as secondary to other conditions or the result of direct systemic infection.

Occurrence.—Pericarditis occurs in fœtal life (Billard, Tardieu, Heiter); Bednar describes cases in newly born infants; it is common in infancy and childhood. Steffen and Baginsky describe a number of cases occurring in infancy. Of 66 cases of pericarditis in children, Baginsky found 20 to occur during the first year of life. The next greatest frequency was between the first and the fifth year.

Etiology.—The majority of cases occur as complications of acute articular rheumatism (Steffen, Friedreich, Bauer, Baginsky), with or without chorea. Tuberculosis and pleuropneumonia rank next as etiological factors. Pericarditis occurs in the exanthemata, scarlet fever, measles, and typhoid fever. It may complicate pertussis, diarrhœal disorders, otitis, meningitis, peritonitis, mediastinitis, or any septic process, such as osteomyelitis. It is also in the newly born infant concomitant with septic conditions. Finally, traumatism may cause pericarditis. The tuberculous form is uncommon before the fifth year of life (Sée).

Bacteriology.—The pyogenic bacteria most frequently found in pericardial effusions, and which play an etiological rôle, are the pyogenic streptococci and staphylococci, the pneumococcus of Fränkel and Weichselbaum, the tubercle bacillus, the Friedländer bacillus, the Bacterium coli, and the Bacillus pyocyaneus (Ernst).

Forms.—There are the same forms of pericarditis in children as in the adult subject. The forms with effusions have, however, a tendency to become purulent, especially in infants and younger children (Baginsky). In these patients, the fibrinous forms result in localized or general adhesions of the two layers of the pericardium and in partial or complete obliteration of the pericardial sac (adherent pericardium).

Morbid Anatomy.—In the mildest forms, there is only a loss of lustre to the serosa in circumscribed or diffuse areas. The fluid in the pericardial sac may be increased in quantity and may contain cellular elements. In other forms, the surface of the pericardium is coated with a layer of fibrin of greater or less thickness. The fibrin may be in the form of bands or of small villous formations. There may be minute hemorrhages on the surface (Delafield). In more pronounced processes the fibrin is in the form of hemorrhagic tenacious masses forming a thick network of strips or bands (cor villosum). The quantity of fluid in the sac varies. The fluid may contain blood.

In the first stage of inflammation, the connective tissue of the pericardium is infiltrated with lymphoid cells and the vessels are filled with blood. After the third day, new vessels appear in the fibrinous exudate on the surface. Fibroblasts, spindle-shaped, spherical, and branching, form a network in this new tissue (Ziegler). Granulation tissue and finally new connective tissue replace the fibrinous exudate, after a period of weeks (productive pericarditis). The so-called opaque areas of thickened pericardium, the maculæ tendineæ seen in adults, are rare in children (Steffen). Adhesions, either localized or general, may form between the two layers of the pericardial sac, causing its partial or complete obliteration.

Tuberculous forms of pericarditis may occur as miliary infiltration of the parietal and visceral layers of the pericardium. There may be serous, serofibrinous, purulent, or hemorrhagic exudate in the sac, or gray cheesy nodules of tubercle tissue may be present in the epicardial and subpericardial tissue (Ziegler, Baginsky).

Myocarditis, circumscribed or general, may occur in all forms of pericarditis. The adhesive forms are complicated with myocarditis.

Symptoms.—Pericarditis in children manifests itself by rational symptoms and physical signs.

Rational Symptoms.—At the bedside, the symptoms of the different forms of pericarditis cannot be divided into classes. Some of the fibrinous or dry forms run an insidious course without giving any marked symptoms of the disease. On the other hand, large effusions may make their appearance without any previous rational symptoms which are characteristic. This is the case in the forms of pericarditis in infants and children, which occur in septic conditions, in pneumonia, empyema, and in the exanthemata. If attention has been drawn to the heart, it will be found that certain symptoms may be traced to the inflammatory process in the pericardium. If the patients have been suffering from endocarditis of rheumatic origin, empyema, or one of the exanthemata, they show the symptoms of grave cardiac disease. They have an anxious facial expression, with marked pallor and cyanosis of the lips. They do not, as a rule, complain of pain. The respirations are markedly increased, as is also the pulse. Older children may complain of pain or uneasiness in the epigastrium. They also show marked dyspnœa and orthopnœa. In infants there are signs of pain on breathing. In some of the fibrinous forms there is fever, but dry forms of pericarditis may run their entire course without it. The purulent forms give a remittent temperature-curve. The pulse is rapid, varying from 120 to 150. In the forms with effusion, the pulse is irregular. If myocarditis is present, the pulse is irregular and persistently high, and there is an accompanying increase in the number of respirations. There is no case on record in which the diagnosis of mediastinopericarditis has been made in a child during life and confirmed at autopsy, nor does the so-called pulsus paradoxus give any assistance, since it is present in other conditions in childhood (Steffen).

Physical Signs.—In pericarditis, there are the physical signs of the dry plastic forms and the forms with effusion into the sac. The signs of the dry pericarditis and those of the first stage of that with effusion are practically identical and may be considered together.

Inspection.—In dry plastic pericarditis and the first stage of pericarditis with effusion there may be no signs to be detected by inspection. There may be an increased impulse, apparent to the eye, over the whole cardiac area to the left. When effusion takes place, little or no pulsation can be made out over the cardiac area when the patient is in the recumbent position. There may be distinct bulging of the cardiac area, varying with the amount of fluid present. No localized apex impulse is visible when the amounts of fluid are large. There may instead be a diffuse pulsation over the area of the apex and toward the sternum.

Palpation.—In dry pericarditis, and in the first stage of pericarditis with effusion, there is a friction fremitus felt over the areas in which the friction murmur is heard. This may be at the apex, at the base, or along the right ventricle close to the left border of the sternum.

The Apex-beat or Impulse, and its Relations to the Chest Wall in Pericarditis with Effusion.—As effusion takes place, it is indicated by certain physical signs relative to the heart apex, and by the line of dulness to the left. Investigations have shown that, when the patient is in the recumbent posture, pericardial effusion first collects at the base of the heart around the great vessels. It next collects over the anterior surface and in the anterior-inferior cul-de-sac of the pericardium (Voinitch). When the patient is *recumbent* the effusion does not necessarily push up the apex-beat. On the contrary, it separates the heart from the anterior chest wall. In moderate effusion the apex-beat may still be felt in the normal position. As the effusion increases, the apex-beat recedes and becomes less discernible and more diffuse, and in large effusion may disappear. This is especially the case, if there is dilatation of the heart or adhesions at the apex. When the effusion is again absorbed, the apex-beat becomes evident in the former situation.

When the patient is *sitting*, the pericardial effusion collects beneath and behind the heart, and, if the heart is not enlarged or held down by adhesions, the apex-beat may at first be *displaced upward*, and will be felt above and to the outside of its normal position. These facts will explain the failure in certain cases of pericarditis, to obtain the displacement of the apex-beat upward. In one of my cases, a boy of six years, suffering from chorea, endocarditis, dilated heart, and pericarditis, the apex-beat was observed in the beginning of the stage of effusion to be located in the sixth space, slightly outside the nipple line. Effusion having occurred, the apex-beat could still be observed in its former locality, but the area of absolute dulness indicating effusion extended beyond the apex, four cubic centimetres to the left of the mammillary line. The effusion disappeared and the apex then corresponded with the line of dulness of the left ventricle.

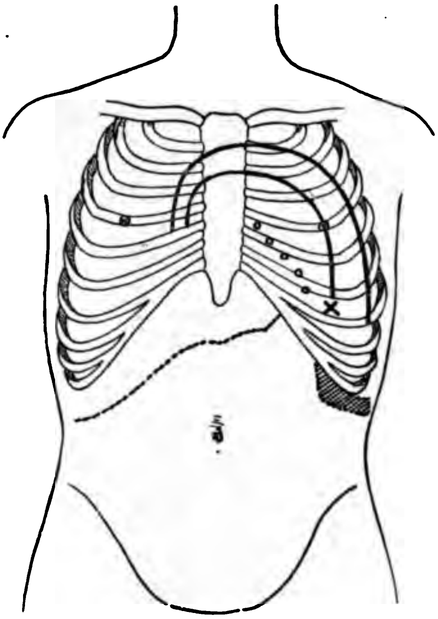
Percussion.—In dry fibrinous pericarditis, and in the dry stage of pericarditis with effusion, there is no increase in the area of cardiac dulness directly traceable to the disease. If there is a slight dilatation or relaxation of the ventricle due to myocarditic complication, the normal præcordial dulness may be more distinct.

The effusion must have a bulk of 40–60 grammes (1½ to 2 fluidounces) before definite signs of its presence can be obtained.

In children, the area of dulness due to pericardial effusion does not have the triangular shape seen in adults. The position of the heart is more horizontal and its shape is retained by the distended sac. Thus, to the left, the dulness may extend in a curved line outside the situation of the nipple. Superiorly, it may extend as high as the first rib. It then extends in an almost horizontal line two or more centimetres to the right of the sternum (Fig. 134). The line of dulness to the right of the sternum then extends downward in an almost vertical line to the liver (sixth space) (Steffen, Baginsky, Ausset). These facts are very important in differentiating dulness resulting from pericardial effusion from dulness due to other causes. Even in moderate effusion there is resistance to the percussing finger. If the patient's position is changed from the recumbent to the sitting posture, the heart falls forward, the pericardial sac is distended, and the dulness to the left may come more toward the mammillary line and, to the right, toward the sternum (Baginsky).

Auscultation.—The friction sound is diagnostic in dry plastic pericarditis and in the first stage of pericarditis with effusion. It may, at the outset, be heard at the apex (Steffen), but is also heard to the left of the sternum over the base, or below, to the left of the sternum, over the fourth or fifth space. Steffen finds it in children, at first, most frequently at the apex. The murmur may be heard on systole or diastole, or on systole only. It may or may not accompany the valvular sounds. It is of very limited distribution, is not conducted, and is of a fine crepitant quality or has a rubbing or a rasping or clicking sound. In the case of a boy suffering from recurrent chorea and pericarditis, there was a loud scraping friction at the apex with murmurs of mitral and aortic regurgitation. I was able in this case to confirm the statement of Walsh, that a loud pericardial friction may sometimes be heard behind, between the scapulæ, to the left of the spine. The friction may for the first day or two be of a crepitant quality and then acquire a rubbing quality. I observed this change in a child four years of age. The patient suffered from dilatation of the left ventricle with mitral insufficiency and stenosis with pericarditis. The friction for two days was crepitant in quality and just audible over the fourth and fifth spaces, to the left of the left border of the sternum. After two days, the

FIG. 134.



Pericardial area of dulness due to effusion in boy, six years of age. Chorea, endocarditis, and pericarditis: *x*, apex-beat before effusion; *o o o o*, friction murmur; outer curved line shows general shape of distended pericardial sac.

murmur of friction acquired a loud rubbing quality. The murmur is sometimes very evanescent or may disappear or reappear at short intervals. The sounds may be intensified by causing the patient to lean forward. When effusion appears, the friction sounds may entirely disappear, or may be heard only in areas around the great vessels or indistinctly over the præcordium. A knowledge of these facts is important in making a diagnosis of fluid in the pericardial sac. The friction sounds may reappear on absorption of fluid. Pleuropericardial friction sounds are rough or fine sounds obtained in children as in adults with the respiratory movements of the lung. They are intensified on expiration and disappear when respiration is momentarily suspended. They may be heard over any part of the præcordium. They are caused by the rubbing of the inflamed pleura and pericardium against each other. This friction is limited to one edge of the cardiac area, generally the left, and is sometimes heard in the back, on the left side.

The **diagnosis** of pericarditis can only be made from the physical signs. In dry plastic pericarditis and the first stages of pericarditis with effusion, the friction sound is the diagnostic sign. If a pericardial friction is once obtained, careful watch should be kept for the appearance of fluid. It is not possible at the outset to differentiate a dry pericarditis which will remain as such, from the first stage of a pericarditis with effusion.

In the stage of effusion, small amounts of fluid will sometimes escape diagnosis. This is likely to occur if a process such as empyema is in progress on the left side. The first stage of a pericarditis may escape diagnosis if the friction sound is evanescent. If the effusion appears in considerable quantity over the great vessels, percussion is made in this region, especially to the right side of the sternum at the level of the second or third space, for an increase in dulness due to a distended pericardium. Absence of dulness in this region across the sternum and for a few centimetres to the right of the right border is presumptive evidence against the presence of any considerable effusion. If dulness exists to the right of the sternum, low down only on a level of the fourth interspace, there is probably no pericardial effusion, but, instead, dilatation of the right ventricle.

Differential Localization by Percussion of Pleural and Pericardial Effusions.—In cases in which pericardial effusion is very large or in which there is pleural effusion into the left side of the chest, a question may arise as to whether there is a simple pleural effusion general or localized, pericardial effusion, or both. Percussion along the sternum will in simple left pleural effusion easily mark out the displaced left pleural fold. Even if there are large amounts of fluid, the fold of the left pleura will be found to be distinctly displaced toward the right border of the sternum. The pleural line will never, except under very exceptional conditions, pass beyond the border of the sternum to the right. If large pericardial effusion is present, the dull note of the effusion extends beyond the right border of the sternum. In left pleuritic effusion the apex of the heart is found by auscultation to be distinctly displaced to a situation beneath the sternum, while in pericarditis it will at first be found to be in the normal position and subsequently to disappear or to be displaced upward and outward.

The **prognosis** of rheumatic pericarditis is good. The purulent forms of pericarditis are in the great majority of cases fatal, especially in very young infants. In older children, I have seen cases of purulent pericarditis, due to infection from a concurrent pneumonia or empyema, recover with timely pericardotomy. The septic forms of purulent pericarditis, complicating sepsis of the newly born and forms of osteomyelitis, are fatal.

The **treatment** of the dry fibrinous forms of pericarditis is limited to the relief of the pain and the treatment of the primary condition, rheumatism. The pain is best relieved by the administration of mild opiates. Codeine in small doses is efficient in many cases. I am not in favor of blistering the præcordial region in children, or of applying a seton, as is done in adults. If the heart is tumultuous, small doses of digitalis in the tincture form and the constant application of an ice-bag over the præcordial region are the most effective remedies. Some authors believe that the ice-bag is also a very powerful means of limiting the inflammation. In rheumatic or choreic cases the salicylate of sodium is given, or if this disagrees with the patient, the ordinary bicarbonate of sodium in doses of grains x (6.5) three or four times daily. Perfect rest in bed, long after the process has run its course, is indicated, on account of the ill effects of strain on the heart affected by myocarditic changes which are undoubtedly present in many of the cases.

When effusion has taken place, the question of the advisability of puncturing and exploring the pericardium always arises. It is very difficult to choose the proper time for entering the pericardium. I have had a number of cases of pericarditis with effusion recover without being subjected to what is at best a hazardous procedure. I can only detail my own practice in these cases. I temporize until the orthopnœa and cyanosis are extreme and evidences of pressure are marked. Too much importance should not be attached to ordinary symptoms. On the other hand, if the temperature is high and daily remits to near the normal, there may be a purulent effusion. If after a reasonable length of time the patient steadily loses ground and the signs of effusion are marked, the pericardium should be entered to determine the character of the exudate. If it is serous, ordinary aspiration will suffice, but if purulent, the operation of pericardotomy should be performed. Pericardial puncture or incision is performed in the same manner as in adults.

It may be remarked that Henoch has never punctured the pericardium. In one of his cases, post-mortem examination showed small sacculated purulent collections of fluid which could hardly have been evacuated by a single puncture. I found a similar condition post mortem in a case in which puncture of the pericardium was undertaken, and resulted in puncture of the heart.

Adherent Pericardium.

Adherent pericardium is an agglutination, localized or complete, of the visceral and parietal walls of the pericardial sac which becomes partly or completely obliterated. The condition follows either a dry plastic pericarditis or a pericarditis with effusion, in the stage of absorption. In the latter case, if the absorption of fluid has been observed and the redux friction-sound obtained, adhesion of the pericardium may be suspected from certain signs; otherwise, diagnosis even within probable limits would in many cases be an impossibility. Infants and children who have withstood an attack of pericarditis, especially of the rheumatic form, are very prone to contract this form of pericarditis. In most cases it causes myocarditis of a progressive type; hence the importance of understanding the condition. Hypertrophy of the heart, atrophy of the heart, or dilatation of that organ may accompany adherence of the pericardium.

The **symptoms**, especially in the rheumatic cases, develop late in the disease when myocarditis supervenes. The condition may prove fatal by progressive affection of the cardiac muscle. One of my cases, of rheumatic origin, showed post mortem no valvular lesion. There were complete obliteration of the sac and extreme dilatation. The symptoms are at first negative. There may be a friction sound or a roughening of the cardiac sounds at the base. There is in some cases a drawing inward of the apex area of the chest at the xiphoid cartilage. A wave-like undulation of the cardiac area with an increase of cardiac dulness is sometimes found. There may be persistent asystole not controllable by digitalis (Sée). In my cases there were angina, a persistently high pulse with an increase in the number of respirations, and in the last stages, all the symptoms of non-compensatory dilatation of the ventricle which are seen in valvular disease. There may be a mitral systolic murmur simulating that seen in valvular disease. In spite of all these symptoms, it is rarely possible to make a positive diagnosis during life.

II. DISEASES OF THE HEART.

The height of the heart and of the great vessels in children does not after the third year materially differ from that in the adult. The ratio of the transverse to the sagittal diameter of the chest in newborn infants is 2 to 1, while in adults it is 3 to 1. This fact should not be forgotten in estimating the size of the heart in infants and children. What in an adult might appear to be a large heart, is normal in an infant or a young child.

Position.—In the first year of life the long axis of the heart is more horizontal than in later childhood or in adult life (Rauchfuss). At the third year, the position of the heart is practically that found in the adult (Dwight).

As the child becomes older the heart assumes more nearly the vertical position, and in older children the apex-beat may be found 0.75 to 1 centimetre within the mammillary line. The situation of the mammillary line is variable in young children; the nipple is over the fourth rib, but further removed from the midsternal line than in older children on account of the greater transverse as compared to the longitudinal diameter of the thorax. In older children the heart areas closely resemble those in the adult. In infants and young children there are certain variations from the adult condition which should be borne in mind.

Size.—The heart is relatively larger in the infant than in the adult, having 0.89 per cent. of the body weight in the newborn infant, while in the adult it has only 0.52 per cent. (Vierordt).

Apex-beat.—The apex-beat in the newborn infant may be felt higher than in the adult. On account of the greater breadth of heart as compared with that of the chest the apex is external to the mammillary line. Steffen says, that normally the apex-beat may be found 1 centimetre external to the mammillary line, or in the mammillary line, or internal to the mammillary line. The apex-beat in infants and children is in the fifth space.

Inspection.—Inspection shows in some cases an undulatory movement over the whole cardiac region. This is normal as long as it is confined to the left of the sternum, but an undulatory movement to the right of the sternum is probably indicative of dilatation of the right ventricle with or without hypertrophy. In rachitis the cardiac region is sometimes unduly prominent. This condition must be distinguished from the more pronounced fulness in the præcordium occurring in cases of hypertrophy or of pericardial effusion. The apex-beat should not be mistaken for an apparent apex-beat which is sometimes seen in young children in whom the intercostal space to the left of the large cardiac dulness is raised with each pulsation of the apex. Percussion in these cases will show the apex to be situated elsewhere to the left and downward. In some cases the apex, instead of pushing the intercostal space forward, draws it distinctly inward. This is in part due to adhesions between the heart, pericardium, and parts external to the pericardium. When children are struggling, the systolic impulse of the heart is seen to be communicated to both the carotid artery and the jugular vein, the vein getting its impulse from its proximity to the artery. The vein may be found to be collapsed and the artery to show an impulse on systole.

Palpation.—The following points may be determined by palpation with the tips of the fingers or full palm:

1. Location of the apex-beat.
2. Sometimes the location of the left boundary of the heart.
3. The force of the systole, hypertrophy or dilatation of the heart, especially if pulsation is evident to the right of the sternum.
4. Transposition of the heart to the right.
5. The closure of the valves of the pulmonary artery in the second or third space near the sternum.
6. Murmurs which cause friction (pericardial) or thrills (endocardial).
7. Rhythm of the heart action.

Auscultation.—In infancy the muscular quality of the first sound is not apparent. The heart-sounds have more the character of the tick-tack of a watch. The muscular character of the first sound fully develops toward the second year of life. All through infancy and childhood there is a natural accentuation of the second pulmonic sound. Too much importance should not be attached to the accentuation even if it is marked.

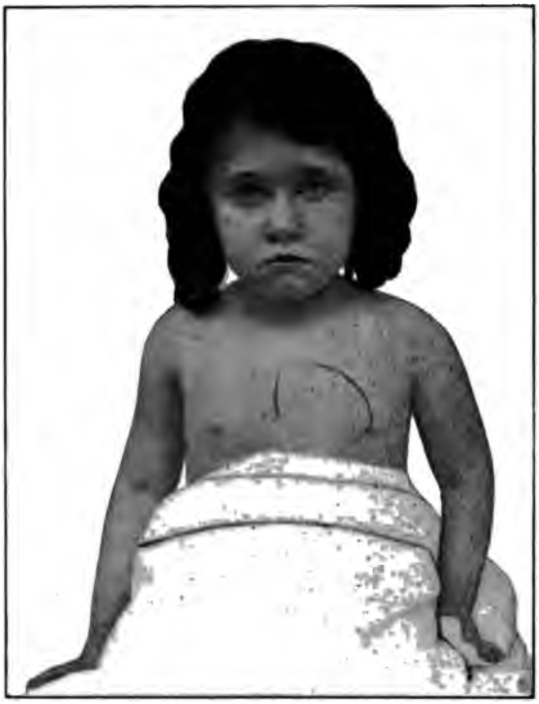
Percussion.—The percussion of the heart has been the subject of much refinement of methods, which only tends to confuse a simple matter. The following method will be found suitable for most clinical purposes:

The lines of demarcation are the midsternal line and a parallel line running through the left nipple. All reckonings as to the limits of cardiac dulness may be safely made from these lines, and such figures will be understood by all physicians. The right border of the sternum is not a good line to reckon from, since the width of the sternum varies. The recumbent posture is preferable in infants; both the recumbent and upright positions are suitable in older children.

Method of Locating the Line of Dulness of the Left Ventricle.—To locate the external boundary of the ventricle, we begin to percuss in the lines parallel with the second, third, fourth, and fifth ribs toward the heart, from the axillary line or the anterior axillary line. To percuss from the midsternal line outward does not in children give as good results.

To locate the external border of the right *ventricle*, we percuss along the *fourth rib* or *fourth space* toward the sternum from the right mammillary line. In young infants a portion of the right auricle and ventricle will be found as high as the junction of the second rib and the sternum (Symington), but it is an ultra-refinement of percussion to try to make out the projection of this part of the right auricle to the right of the sternum. It is found, anatomically, that the curve of the auricle to the right of the sternum begins at the third space, and is most marked behind the fourth costal cartilage. It is sufficient for clinical purposes to make out this most projecting part of the heart to the right of and behind the sternum.

FIG. 135.



Form of the normal relative cardiac dulness in a child two and one-half years of age.

The apex of the heart is generally made out by percussing along the fifth rib or fifth space from the antero-lateral axillary line toward the midsternal line. The external boundary of the left ventricle is in children slightly outside the apex-beat. The area of cardiac dulness which is absolute and which is uncovered by lung can best be made out by percussing from above downward over the cardiac area. In children or infants this area cannot be marked out as definitely as in the adult. The younger the child or infant, the greater the difficulty. In infants and children interest centres rather in the apparent size of the heart (relative dulness) than in the area uncovered by lung.

The dulness extends to the right and left of the midsternal line, at a level with the fourth rib, as is indicated by the following figures compiled from Steffen's tables:

Infants under one year	right v. 4 to 6.5 cm. to right. left v. 3.5 to 6.25 cm. to left.
Children one to two years	right v. 4 to 6.5 cm. to right. left v. 4 to 7.25 cm. to left.
Children two to three years	right v. 4.5 to 7.5 cm. to right. left v. 4.5 to 6.5 cm. to left.
Children five to six years	right v. 5.5 to 7.25 cm. to right. left v. 5 to 8.25 cm. to left.
Children nine to ten years	right v. 5.5 to 8.5 cm. to right. left v. 5.5 to 8.5 cm. to left.

Enough has been selected to show that the actual size of the heart as obtained by percussion in infants and children is extremely variable, and the examiner must be guided by the relative size.

Congenital Heart Disease.

Congenital heart disease may be suspected from certain physical signs which occur in that condition and are in a sense characteristic of it. These are cyanosis, changes in the area of cardiac dulness, and the presence of characteristic murmurs.

Cyanosis.—The cyanosis which is characteristic of congenital heart disease does not occur in any of the acquired cardiac lesions. It is most common in the congenital forms of pulmonary stenosis of the artery, conus, or ostium. On the other hand, it may be absent in marked congenital disease, as in deficient ventricular septum and open ductus arteriosus. In the latter disease it may appear late in the condition, only at intervals, or not at all. It may be absent at birth and appear in infancy or childhood.

Cardiac Dilatation and Hypertrophy.—The presence of a murmur of congenital origin does not necessarily indicate change in the area of cardiac dulness. In fact, a normal cardiac area is sometimes evidence of the congenital character of a murmur. Hypertrophy of the left ventricle should be present with hypertrophy of the right ventricle, and a murmur to indicate open ductus arteriosus. Dilatation of the right ventricle is of value when present with a murmur indicating stenosis at the pulmonary valve. On the other hand, marked congenital defects may exist without any change in the size of the ventricle. Moreover, if the cardiac area is enlarged and the apex impulse weak, congenital disease may be suspected. The weak apex impulse indicates dilatation.

Murmurs.—The murmur most characteristic of congenital heart disease is a systolic murmur at the situation of the space between the second and third costal cartilage to the left of the sternum, and not conducted into the arteries of the neck. It is only when there are complicated defects that murmurs are conducted into the carotids (open ductus arteriosus).

Fœtal endocarditis affecting the tricuspid or *mitral valves* is rare, and therefore murmurs of congenital origin are rare at these valves.

Diastolic murmurs are, so far as congenital lesions are concerned, of theoretical interest only.

Systolic murmurs, such as those heard in cases of defects of the ventricular septum, and which cannot be attributed to valvular disease, occur at the pulmonic valves. In these cases the murmur has no point of greatest intensity, but is heard not only at the valve, but also over the whole præcordium. The valvular sounds are distinct. The most marked congenital defects or disease of the heart may exist without any murmur or other physical signs during life.

In simple pulmonary stenosis, the second pulmonic sound is weak; in cases complicated with *open ductus arteriosus and hypertrophy of the ventricles*, it is accentuated; in cases of pulmonary stenosis and deficient ventricular septum, it is either weak or very low.

The positive diagnosis of the exact lesion in congenital heart disease is in many cases impossible. The reason for this is easily found in the fact that if the patient lives longer than the first year, the lesion is rarely simple, but occurs with other congenital defects in the heart. Another cause is the rarity of autopsies on uncomplicated cases which have been carefully studied during life. Lastly, in complex cases, even if the diagnosis has been confirmed at autopsy, it is impossible to say to what degree the lesion diagnosed and the other complicating conditions found at autopsy have been the cause of the signs and symptoms found during life. The physical signs of congenital heart disease vary as the lesion is a simple one or is combined with other congenital defects. The following classification of congenital heart disease of developmental or fœtal endocarditic origin will be found useful in clinical work:

1. *Septum Defects.*—Auricular (foramen ovale); ventricular.
2. *Pulmonary Artery.*—Stenosis of the conus, trunk, or ostium: (*a*) simple cases (before the end of the first year of life); (*b*) complicated cases with open foramen ovale or ductus arteriosus, defect of the ventricular septum, or transposition of the great vessels.
3. *Aortic Valve Stenosis or General Contraction of the Aortic System.*—The first may be due to developmental defect or to fœtal endocarditis; the second, to developmental defect. All aortic conditions anomalous in character have, so far as is known, not been positively diagnosed during childhood.
4. *Valvular anomalies* of the semilunar valves, due to fœtal endocarditis or developmental irregularities are of purely scientific interest.

5. *Open Ductus Arteriosus or Botalli.*—(*a*) Simple; (*b*) combined with septum defects or pulmonary stenosis.

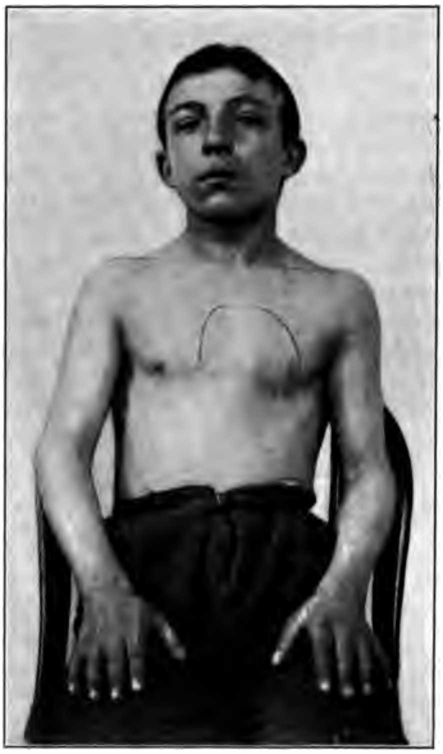
6. *Transposition of the Heart and Congenital Anomalies of the Pericardium* (of purely scientific interest).

From the above account, which I have modified for practical use from the classification of Vierordt, it will be seen that only the congenital anomalies of the auricular ventricular septum, the pulmonary artery, and the ductus arteriosus Botalli are of interest to the clinician.

Stenosis of the Pulmonary Artery, Conus, or Ostium.

This is the most common of all congenital heart lesions. If found after the thirteenth month of life, it is in most cases combined with a congenital deficiency of the septum ventriculorum. Rauchfuss found a simple stenosis in only 10 per cent. of all the published cases. Most of the cases are due to fœtal endocarditis.

FIG. 136.

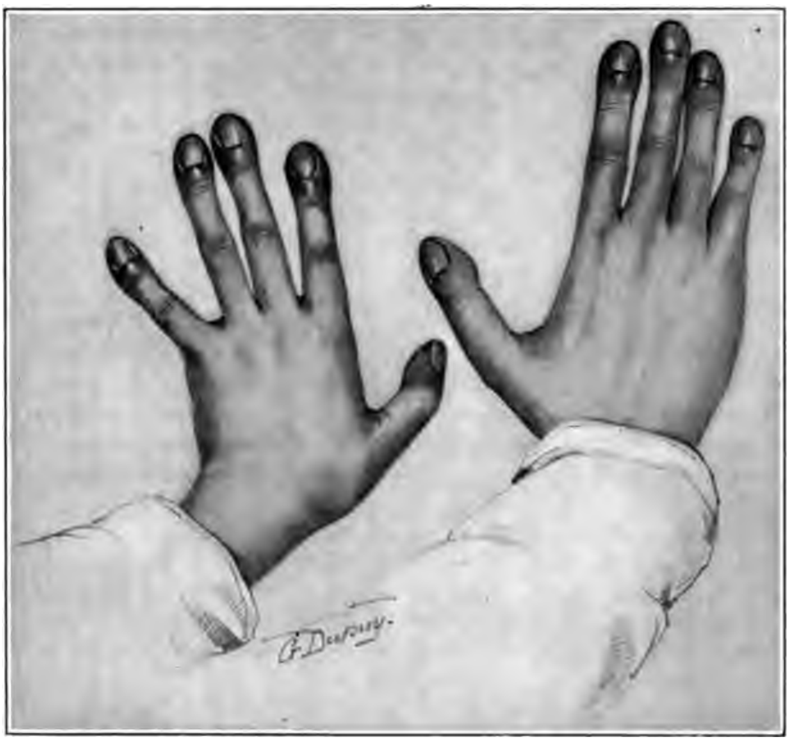


Congenital pulmonary stenosis with open ductus Botalli, as shown by a dull area in the second space above the base of the heart; loud systolic murmur at the pulmonary orifice; clubbed fingers, cyanosis of the general surface, symptoms of chronic bronchitis; dyspnœa on exertion. Boy, twelve years of age.

Physical Signs.—**Simple stenosis** of the artery, conus, or ostium, found only before the thirteenth month (Rokitansky).

Cyanosis.—Early and congenital cyanosis and signs of venous stasis, such as clubbed extremities of the fingers, even in young infants. In cases which are met in later life the clubbing of the extremities of the fingers and cyanosis of the finger-tips are marked.

FIG. 137.



Clubbed fingers of congenital heart disease. Child six years of age.

Murmur.—A systolic murmur heard with greatest intensity at the situation of the pulmonary valve to the left of the sternum, between the second and third costal cartilages, and not conducted into the carotids. A weakened second sound at the pulmonary valve; dilatation of the right ventricle.

Simple stenosis is found in infants, but is rare. In most cases there are also present congenital defect of the ventricular septum, open ductus arteriosus, tricuspid changes, or the aorta arises from the right ventricle or both ventricles. The following facts should be kept in mind in the diagnosis of cases occurring after the thirteenth month of life:

If the above signs are present with a weakened second pulmonic sound, there being absolutely no conduction of the murmur into the carotids, it may be assumed that there is a pulmonary stenosis with an open foramen ovale.

Conduction of the murmur into the arteries of the neck, with a very distinct though not accentuated second pulmonic sound, points to the presence of a septum defect with a pulmonary stenosis.

An accentuated second pulmonic sound with conduction of a murmur of a loud buzzing character into the subclavian and carotids, and a hypertrophy of the right and also of the left ventricle, will support the theory of a pulmonary stenosis with a patency of the ductus arteriosus (Fig. 136) (Hochsinger). In these cases of open ductus arteriosus there is a thrill and a distinctly defined area of dulness in the second space to the left of the sternum above the base of the heart. This dulness is of great diagnostic import. It is due to the dilated great vessels at the base of the heart.

As an exception to the above classification, may be mentioned the case of Sansom, in which cyanosis and extreme anæmia were present. In rare cases, the second pulmonary sound may be very low. The murmur may be conducted into the axilla, the right heart not being dilated.

Open Ductus Arteriosus or Ductus Botalli.

This is a very rare congenital defect. There are in the literature only 20 cases of uncomplicated open ductus arteriosus in which the autopsy confirmed the clinical diagnosis. Of these, only 5 occurred in infants under one year of age, and 5 others ranged from the first to the tenth year (Vierordt). The complicated cases occur with stenosis of the pulmonary artery, septum defects of small extent, and open foramen ovale.

Physical Signs.—**Cyanosis** is not present in the majority of cases, or if present is so only intermittently and is not marked.

The **murmur** is a loud buzzing systolic murmur heard with greatest intensity over the pulmonary artery, and not conducted downward, but conducted to the left of the sternum into the veins of the neck (Hochsinger).

There is an accentuated second pulmonic sound which can be heard in the *carotids.*

Right Ventricle.—The presence of hypertrophy of the right ventricle tends to confirm the diagnosis ; if the left ventricle is also hypertrophied, greater certainty is added. This is of great moment, since hypertrophy of the left ventricle is not present in any of the other congenital defects, except those connected with the anomalies of the aorta and aortic system and which have only a scientific value, since the literature contains no cases which have been diagnosed during life. The dulness in the second space referred to under Pulmonic Stenosis is also of value.

Congenital Defects of the Auricular Ventricular Septum; Defects of Auricular Septum; Open Foramen Ovale.

Inasmuch as 44 per cent. of the autopsies upon individuals who during life showed absolutely no signs of cardiac disturbances reveal a patency of the foramen ovale, the diagnosis of the condition as an uncomplicated entity should be made with great reserve. This congenital defect is generally found to exist in connection with other defects of a congenital nature (stenosis of the pulmonary artery).

Cyanosis has been found in all the cases in which autopsy has been made. In a case recorded by Foster, there was cyanosis with a varying systolic and presystolic murmur at the sternal end of the third or fourth costal cartilage.

Walshe says that it can hardly be asserted positively that a patency of the foramen ovale may of itself cause a murmur.

Congenital Deficiency of the Ventricular Septum—Maladie de Roger.

Autopsies have shown that this condition may exist during life without giving any signs of its presence. Moreover, it is so often combined with other congenital heart anomalies, such as stenosis of the pulmonary artery or ostium, that the signs of the ventricular condition must of necessity be obscured by those of the complicating defect.

Cyanosis has been present in some cases of uncomplicated ventricular septum defect (Müller) and absent in others. It is present in the cases complicated with pulmonary stenosis.

Murmur.—According to Roger, a loud systolic murmur is heard over the whole præcordium, toward the median line, over the upper third of the cardiac area. According to others (Müller), *the murmur has no special point of greatest intensity.* It is not conducted into the vessels of the neck.

Rauchfuss calls attention to the fact that with this murmur the distinct valvular character of the heart-sounds at the various valves should be heard. The case of Müller was that of a cyanotic infant two months old. A loud murmur having no special point of greatest intensity was heard over the whole cardiac area. The valvular sounds were distinctly heard. Autopsy showed uncomplicated defect of the ventricular septum.

Acute Endocarditis.

Acute endocarditis is an inflammation of the lining membrane of the heart. That covering the valves and their immediate vicinity is the area generally affected. There is also an inflammation, slight or marked, of the muscle tissue of the heart, and in some cases there is inflammation of the pericardium. Endocarditis thus involves structures of the heart other than the endocardium. Acute endocarditis may be benign or malignant. Between the two extremes, there are all gradations as to severity. All forms of endocarditis are caused by infection which in the malignant variety is of the severest septic type. Fœtal endocarditis affects the right side of the heart; after birth, the left heart is chiefly affected. The condition is less frequent before than after the fifth year of life, and occurs with equal frequency among boys and girls (Steffen).

Etiology.—Acute endocarditis occurs most frequently with acute articular rheumatism, but may appear in any infectious disease. It is often found in scarlet fever; less often in measles. I have seen it in rare cases of erythema nodosum (2 cases). It may occur with typhoid fever, diphtheria, influenza, pneumonia (Netter), cerebro-spinal meningitis, and tuberculosis. In fact, all forms of sepsis, such as osteomyelitis, either fœtal or in the newborn infant or in children, may be accompanied by endocarditis. Endocarditis is present in 16 per cent. of the cases of chorea and is always present in fatal cases of that disease.

Bacteriology.—The most important bacteria bearing an etiological relationship to endocarditis are the streptococci of the various varieties and the Staphylococcus pyogenes. Harbitz divides endocarditis into the infectious and the non-infectious varieties. He found bacteria in the vegetations in most of the infectious cases, streptococci in 39.5 per cent. and staphylococci in 18.6 per cent. of the cases; other bacteria, such as the pneumococci, were also found. The cases in which no bacteria were found were healed cases. He thinks that the staphylococci most often cause pyæmic endocarditis with ulcerations and metastatic abscess. Welch has, however, found streptococci in ulcerative endocarditis, and does not fully accept the view of Harbitz. The Diplococcus pneumoniæ is next in importance as an etiological factor. Wright found the Bacillus diphtheriæ in one case. Other bacteria, such as the Gonococcus, the Bacillus endocarditidis griseus (Weichselbaum), the Micrococcus endocarditidis rugatus and capsulatus, the Diplococcus tenuis (Klemperer), have been found in cases of adult endocarditis. Although they are all, as well as the Bacillus typhosus, doubtless capable of causing the same process in children, actual clinical cases are still to be published.

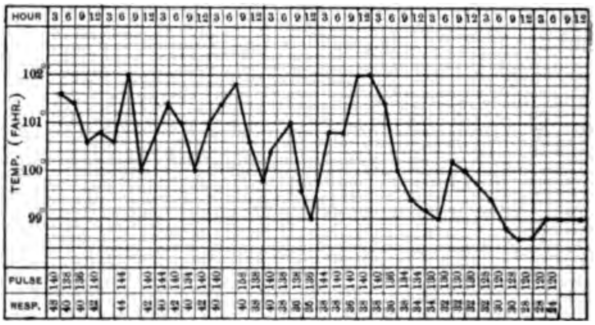
All forms of endocarditis are thus septic processes due to the circulation in the blood of bacteria or their toxins. In some cases it is possible to discover the point of entrance of the bacteria into the circulation, in others, it cannot be fixed upon. The forms of endocarditis are not so uncommon in infants as is supposed. The tonsil is a great avenue for the entrance of bacteria or toxins into the circulation (Cheadle). It is believed that many cases of endocarditis in children originate in this manner (Packard). I have frequently met with endocarditis in which the only other clinical manifestation was a slight redness or swelling of the tonsils. The integrity of the endothelium of the endocardium must be compromised if bacteria have invaded the tissue of the valvular endocardium (Prudden). It is supposed that the toxins produced by the bacteria circulating in the blood reduce the resistance of the endothelial lining of the endocardium, thus preparing the soil for bacterial invasion.

Morbid Anatomy.—In some cases the only lesion is a swelling of the valves. They are thickened and succulent, their surface being smooth. The basement substance is swollen and there is an increase of connective-tissue cells (Delafield). In other cases the borders of the valves present transparent, gelatinous, whitish-yellow or reddish formations, varying from the size of a pin's head to that of a bean. These are irregular in shape, cover both surfaces of the valves, and may be single or multiple. They are also seen on the chordæ tendineæ. The free border of the valve is warty or papillomatous (endocarditis verrucosa or polyposa) (Ziegler). The papillæ may appear on the free surface of the valves. There may be a loss of substance with the formation of adherent thrombi of a whitish or reddish color and of tenacious consistency (endocarditis ulcerosa). Small foci of pus may be present in the heart substance (endocarditis pustulosa). Bacterial invasion of the surface of the valves results in loss of substance, formation of thrombi, and changes in the nuclei of the connective tissue (necrobiosis). The mitral valve being more vascular is sooner affected than the aortic or pulmonary valves. Exudation on the valve is replaced by new connective tissue; excrescences and new formations become permanent. If the bacteria penetrate deeply, thickening of the valve results. Large thrombi are organized, and the valves become shrunken and distorted. Ulceration and loss of substance may result in perforation of the valves. The thrombi just mentioned are sometimes made up of blood-plates; in other cases leucocytes, blood-cells, and fibrin in varying amounts are present.

There may be exudative pericarditis. The myocardium is the seat of degeneration, which leads to dilatation, or to abscess or aneurism of the heart muscle. Through the separation of portions of the thrombi or of the vegetations on the valves, these particles may be carried into the circulation. Containing, as they do, bacteria (mycotic emboli), they cause secondary infections with necrosis or abscess in the kidney, spleen, and brain.

The **symptoms** of acute endocarditis are those of some general infection. They are not in infants and children so characteristic as to direct attention to the heart. Infants cannot and children do not complain of pain, palpitations, or feelings of uneasiness in the heart region as adults sometimes do, and therefore unless the heart is carefully examined as a routine procedure, the simple cases of endocarditis will escape observation. The most interesting cases are those which begin with all the symptoms of an attack of influenza or tonsillitis. There are fever, rapid pulse, and an increase of the respirations to 36 or 40. The fever, however, does not subside in the time occupied by the course of one of the above affections; it continues high, 103°–104°–105° F. (39.4°–40.5° C.), with morning or afternoon remissions. In such cases a most careful examination of the lungs and other organs, fails to reveal anything abnormal. The heart, however, shows the presence of endocardial inflammation. In some obscure cases, there is an increasing pallor with a slight daily rise of a half a degree or a degree in body temperature, which will continue for days or even weeks and give rise to a suspicion of paludal poisoning. There is also an increasing pallor. Examination of the heart reveals the lesion. In other cases there are a very slight but increasing pallor, weakness, and indefinite pains in the bones and joints. In children, more than in the adult subject, we are apt to have monarticular affections of a rheumatic nature. I have seen several cases of monarticular joint-affection with an endocardial murmur in childhood. One case was that of a child two years and eight months of age, another recent case was that of a child eight years of age. In the one case the ankle was swollen, painful, and slightly reddened. There was no temperature. There had been slight pain in one knee some days previous to the ankle-affection.

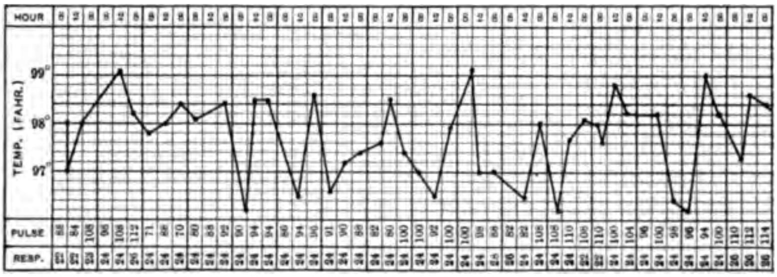
Fig. 138.



Endocarditis complicating influenza. Second week of the illness. Mitral systolic mumur developed under observation. Female child, four years of age.

In the other case the metatarsal and phalangeal joint of the small toe was involved. In young children the joints may be painful, and still no history of joint-pain will be given, and the first indication of pain is a decided limp in walking. The rheumatic cases are as a rule easily diagnosed. The heart should be regularly examined in such cases. The endocarditis which complicates chorea sometimes runs its entire course without any marked rise in the body temperature. I have, however, been able in such cases to confirm the statement of Jürgensen, that the normal diurnal temperature variations are distorted—that is to say, the morning temperature may be higher than the evening temperature. In other cases of chorea there is a distinct rise of temperature without any increase of the respirations and pulse-rate during the active stage of the endocarditis. After the symptoms of chorea have begun to decline there is occasionally a rise of temperature lasting a day or more, which may indicate a slight recurrence of the endocarditis. In other cases I have observed a subnormal temperature of a degree or more lasting for days. This occurred in a case of recurrent endocarditis. Thus the temperature is not at all characteristic. The heart in children is extremely irregular. It may vary from 60 to 120 per minute within a few days, and may vary at different times of the same day. Under such conditions it may be surmised that there is a myocarditis. The respirations are increased. The children do not complain of the heart.

FIG. 139.



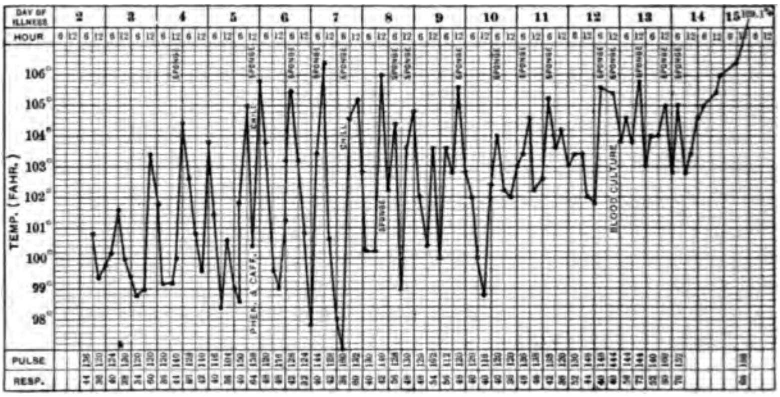
Chronic cardiac disease, hypertrophy, and dilatation of the left and right ventricles. Enlarged liver and spleen, ascites, cyanosis, recurrent attacks of endocarditis. Temperature by rectum shows a subnormal range. Boy, twelve years of age.

In pneumonia, scarlet fever, and measles, the endocarditis is masked by the symptoms of the primary disease.

Septic, Ulcerative, or Malignant Endocarditis.

This form of endocarditis is rare in infants and children. Adams collected from the literature 47 cases in children. The sexes were about equally affected. Three cases were congenital and 8 were five years of age or under. The others ranged up to fourteen years. The trend of opinion (Adams) supports the contention of Lazarus, Barlow, and Weichselbaum, that these cases differ from the benign cases only in regard to severity. Dreschfeld divides these cases into the following classes: (*a*) the primary form, (*b*) the form complicating septic disease, (*c*) the form complicating pneumonia and meningitis, (*d*) the form which occurs as a mixed infection due to septic organisms in the acute infectious fevers or which is secondary to the rheumatic affections of the valves. I have recently observed two cases of septic endocarditis. In one, in a boy with osteomyelitis of the tibia, staphylococci were found in the blood during life. In the other case, which followed a pneumonia, streptoccoci were found in the blood during life. In the former case hemorrhagic symptoms and signs of severe cardiac disease, such as gallop-rhythm, were observed. The latter case was seen in my hospital service. The child, a girl of eight years, had had a pneumonia three weeks previous to her admission. She had apparently recovered, had sat up in bed after ten days, and was about. A day before her admission the temperature mounted to 104° F. (40° C.), she vomited, and had diarrhœa. The child showed much prostration, and on examination an area of consolidation was found in the right lung behind. She had an active endocarditis giving a mitral systolic murmur. The liver and spleen were large; the temperature rose and fell twice daily, chills and dyspnœic attacks preceding each rise. The temperature subsided to the normal or subnormal after each rise. There were nausea, vomiting, and signs of cardiac failure. The heart did not at first show any enlarged area of dulness. After a few days the left ventricle showed an increased area of dulness to the

FIG. 140.



Fatal septic endocarditis following a pneumonia. Streptococci found by culture in the blood during life. Girl, eight years of age.

extent of 2 to 3 centimetres outside the nipple-line (acute dilatation), with diffusion of the apex-beat. The right ventricle was dilated. With the extreme fluctuations of temperature, the child became delirious. The heart, as at the time of admission, showed a mitral systolic murmur. After ten days petechiæ appeared, first on the neck and upper thoracic region, and increased both in number and extent. The face and eyes became œdematous (cardiac failure). The patient became unconscious and died in coma with Cheyne-Stokes respiratory phenomena. The blood withdrawn during life showed in culture the presence of long streptococci.

The **diagnosis** of septic endocarditis rests on the history and the presence of cardiac signs, the prostration, the great fluctuations in temperature resembling those in sinus thrombosis in ear disease, the onset of chills and delirium, the presence of petechiæ, and lastly on the results of examination of the blood for bacteria.

Of great interest in this connection, are the cases of chronic recurrent endocarditis which toward the close of the disease have certain symptoms resembling those of the septic or so-called malignant cases. In a child of ten years suffering from chronic recurrent rheumatic endocarditis, there was toward the close of the illness a period during which phlebitis with thrombosis of the deep veins of the neck and arms on both sides and œdema of the corresponding extremities developed successively. After a few weeks the symptoms of phlebitis and thrombosis gradually subsided and there was a period of a few weeks during which the patient was much improved. The fever and anasarca subsided and the heart action was good. Before the fatal issue the endocarditis recurred and there were fever and what appeared to be significant petechiæ on various portions of the body. The case was a rheumatic one and had been under observation for two years. Its outcome gives weight to the theory that a seemingly benign endocarditis may at any time take on a malignant or septic nature.

Physical Signs of Acute Endocarditis.—A murmur which develops while a child is under observation is indicative of acute endocarditis.

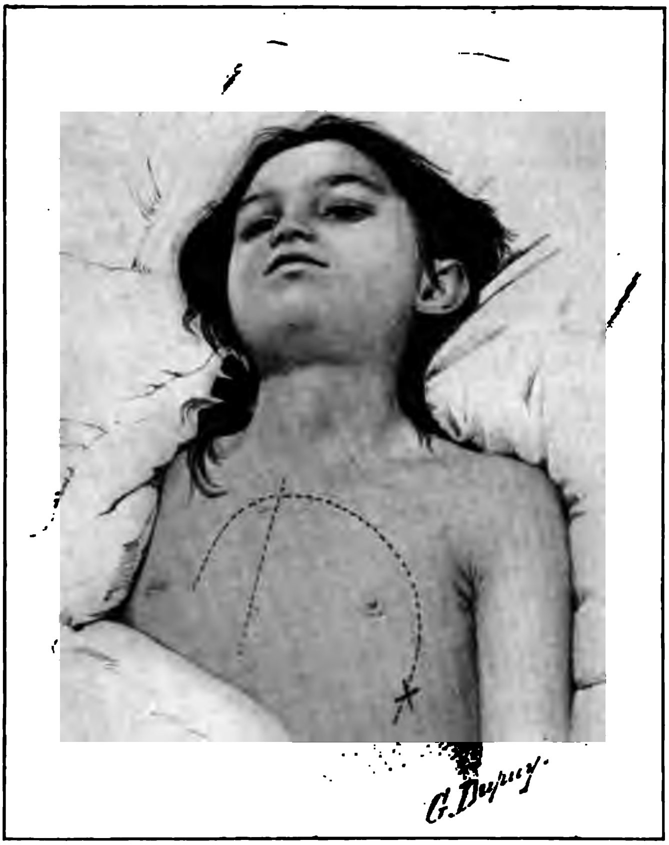
Inspection may reveal nothing abnormal, or there may be extreme irregularity of the action of the heart. There may be increased action, as evinced by visible pulsation over the cardiac area.

Palpation also may reveal nothing abnormal; there may be a thrill over the apex.

Percussion at first reveals nothing. In some cases there is a slight dilatation of the left ventricle (Steffen) as the disease progresses. I have seen this dilatation in cases in which the condition had existed for a week. During convalescence the dilatation may retrograde and the heart confines return to their normal limits.

Auscultation.—In the majority of cases, a soft systolic murmur is heard over the apex and the mitral area. There is rarely a presystolic murmur. There may be murmurs at the other valves, having the characteristics of the same murmurs in the adult. In any acute disease, the physician should be careful to observe a murmur very carefully before pronouncing it organic. I have found murmurs, especially in typhoid fever in young and older children, which appeared and disappeared. Such murmurs are hæmic or myocarditic and functional; they are very gentle, generally systolic, and are limited very closely to the apex or pulmonic area. They are not conducted and there are no positive signs of dilatation. Jacobi has described pulmonic murmurs in very young infants, which were at

Fig. 141.

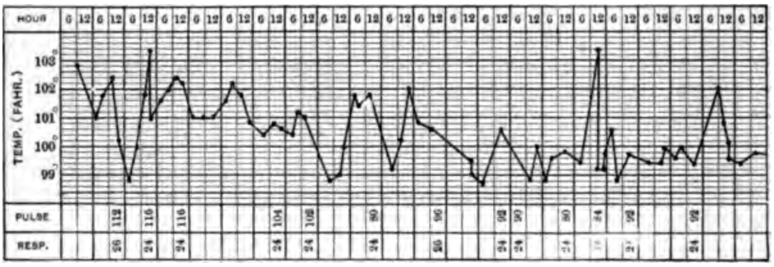


Chronic cardiac disease; great cardiac dilatation; recurrent attacks of endocarditis; phlebitis and thrombosis of the deep veins of the neck and arm on both sides successively; œdema of the corresponding arm and forearm; great dilatation of the superficial cervical and thoracic veins. Female, ten years of age.

autopsy shown to be functional. On the other hand, if a murmur is distributed over a valvular area, takes the place of the valvular sound, is conducted into the arteries, and occurs in connection with signs of dilatation, the physician is justified, acute symptoms being in evidence, in assuming the presence of organic disease.

Course and Prognosis.—Many cases of endocarditis, especially those not of rheumatic origin, run their course, do not recur, and in after-life give no symptoms referable to the heart. Others run an acute course without developing any physical signs until convalescence. I have seen such forms follow chorea. The murmur develops in the intervals of freedom from symptoms of chorea. Rheumatic cases are likely to recur, and in this tendency lies the danger. The prognosis as to immediate recovery is very good in all of the ordinarily severe cases of acute endocarditis. The severer septic or malignant cases give a grave prognosis. The future of cases of acute endocarditis which have recovered will depend very much on the immediate management. I have seen patients who had been allowed to be up and about too early and to participate in sports, develop after a few months symptoms resembling those seen in acute dilatation due to heart strain. These cases show a marked dyspnœa on exertion and cyanosis after play. The children are easily fatigued. They have pain and uneasiness over the region of the heart after running. On percussion an abnormally large heart area is found.

FIG. 142.



Recurrent endocarditis with acute articular rheumatism which developed under observation. Boy, twelve years of age.

The **treatment** of acute endocarditis is directed toward limiting the damage done by the disease to the heart. Rest in bed is necessary. The patient should not be allowed to maintain the sitting posture, but should be recumbent. The rest should be continued long after the subsidence of the active symptoms. The symptoms and physical signs are the guides as to its duration. If there have been marked disturbance of the heart action and distinct dilatation of the ventricle with signs of myocarditis such as great irregularity of the pulse, the stay in bed should be prolonged for weeks.

If the action of the heart is rapid and tumultuous, an ice-bag should be placed over the cardiac area. This remedy is also useful in cases in which the heart action is not very rapid, but in which there are nevertheless signs of active inflammatory disturbances.

Salicylate of sodium is a favorite remedy, not only in cases with a rheumatic history, but also in septic cases. The dosage is grains j to ij (0.6–0.12) every few hours for infants and young children; older children receive more. Some children have stomach pains and disturbances after taking salicylates. There must then in the rheumatic cases be substituted some alkali, such as bicarbonate of sodium. I have administered aspirin in many cases with apparent benefit. A few drops of the tincture of digitalis will be useful in regulating the heart action late in the disease. Digitalis is given for periods of a few days and then suspended for a time, after which it may again be given if necessary. Care should be taken to support but not to drive the heart. The diet should be light, fluid, and easily assimilable. The bowels are best regulated with some saline cathartic or by rectal enemata.

The temperature, if high, may be treated in the same way as in other acute diseases. Baths of low temperature should not be given. The temperature in this disease is of so short duration that in the majority of cases sponging with cold water is effective. The management of choreic cases will be discussed in the section on Chorea.

The injection of antistreptococcic serum in the septic cases has not given satisfactory results.

Chronic Valvular Disease of the Heart.

The lesions in chronic valvular disease in infancy and childhood are the same as in the adult subject.

The **etiology** has been considered in the section on Endocarditis.

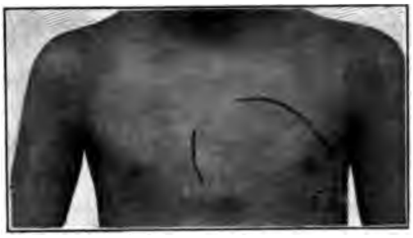
Frequency.—Of 70 of my cases of chronic valvular disease, 37 were of the female and 33 of the male sex; 2 were below the age of two years; 24 from the second to the fifth year, and 39 from the fifth to the tenth year of life. In 50 of the 70 cases the mitral valve was involved, causing either a systolic or a diastolic murmur, or both. The following table will give an idea of the relative frequency of the valvular lesions:

Mitral insufficiency	26 cases.
Mitral stenosis	6 "
Mitral insufficiency and stenosis	18 "
Aortic stenosis	6 "
Aortic stenosis and insufficiency	1 case.
Endocardial and pericardial disease	5 cases.
Combined lesions of mitral and aortic valves	8 "

The **physical signs**, the reservations noted in the section on cardiac murmurs being made, are the same as in the adult subject.

On the other hand, certain characteristics of the disease in childhood are not common to the adult subject. There are cases of chronic cardiac disease in infancy and childhood which escape recognition because the heart is not examined with sufficient care. Murmurs of mild intensity pass unrecognized.

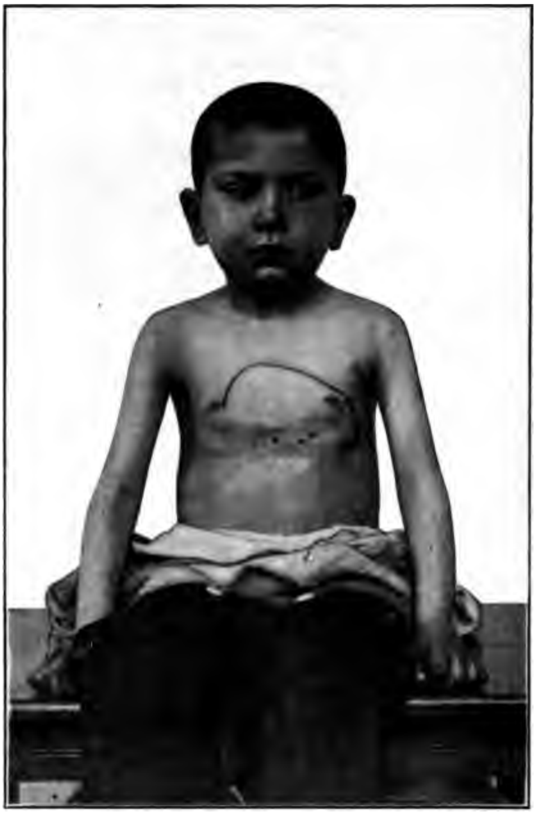
FIG. 143.



Simple mitral insufficiency: dilatation of the left ventricle. Girl, six years of age.

There are cases of endocarditis which run an obscure course, give very few symptoms, and which are apt to recur at the onset of tonsillitis or an attack of influenza. These cases of chronic endocardial disease give very few symptoms in the intervals between the attacks. There may be obscure pains in the limbs or joints which are not interpreted by the physician as purely rheumatic, but are believed to be of a grippal character. The patients may eventually develop symptoms of serious cardiac insufficiency. The cases of chronic valvular disease resulting from an attack of some infectious disease may leave the heart little compromised. It is true that upon examination a cardiac murmur which may be marked or slight is heard, but the cases have no subjective symptoms. They have what is called by the German School a healed endocarditis. They may, however, develop serious cardiac symptoms at the onset of an infection of the gut or other organs. The heart in these cases may be called irritable. The patients do not develop inflammation of the endocardium or pericardium as do the rheumatic cases. On slight disturbance of the gut or intestines, such a heart, even when there is no fever, acts very much like a hypertrophied organ. There is an increase not only of the frequency, but also of the force of the heart's impulse. The vessels are also affected, and there is a bounding full pulse at the radial. As a result of the infection and of the congestion brought about by the increased action of the heart, there will be albumin and casts in the urine. These symptoms subside and do not recur except at long intervals. In the intervals, with the exception of a valvular murmur, there are absolutely no signs of cardiac disease. In children, cases with a slight or marked valvular lesion which are apparently at a standstill, give certain symptoms which are significant of defective cardiac action. On exertion, the children complain of pain in the side or the epigastrium. Examination will show little change in the cardiac areas. The valvular murmur is heard. Such hearts are also irritable. I have often found a distinct history of palpitation occurring at intervals and even in the absence of exertion. Many children with chronic cardiac disease of a very mild and absolutely quiescent type, exhibit a persistent pallor which does not yield to drugs. Children without other symptoms complain of headaches after slight excitement. Examination will, in these cases also, show a slight hitherto unrecognized chronic cardiac valvulitis. Slight œdema of the eyes which is persistent should direct attention to the heart.

Fig. 144.



Chronic cardiac disease; dilatation of the right and left ventricles. Epigastric pulsation. Boy, six years of age.

Many cases without any other signs of chronic cardiac disease show a slight evanescent trace of albumin in the urine.

There may be absolutely no signs of cardiac insufficiency or change in the physical character of the organ. Children with signs of quiescent cardiac disease often have obscure attacks of faintness and vomiting, following every little excitement.

There are also the rheumatic recurrent cases of endocarditis in childhood. These exhibit very much the same symptoms of cardiac insufficiency as the corresponding cases in adults, viz., enlargement of the liver and spleen. Children appear to recuperate more rapidly than adults, but, on the other hand, the attacks are more likely to recur in them than in older subjects. A compromised heart in a child will bear more strain than in an adult. Cases are frequently seen in which children show on physical examination marked chronic disease, but are notwithstanding exceedingly active and show no symptoms referable to the heart. The signs of insufficiency of the cardiac muscle are the same in children as in the adult. There is dyspnœa on exertion, slight œdema of the general surface, and enlargement of the liver and spleen. In the later stages, there are transudates in the pleura and abdomen. In some cases, especially where there is progressive interstitial myocarditis with adherent pericardium, the pleura may show unilateral transudate.

In cases of cardiac insufficiency, the pulse is persistently high or very irregular. There is persistent dyspnœa. Children with cardiac disease suffer, as a rule, less than adult subjects.

Cardiac angina is not an uncommon symptom in cases of aortic disease. It is present in cases in which there are signs of lack of compensation. The angina comes on in attacks occurring chiefly at night, and is very severe. I have seen a boy of eight years with an aortic murmur suffer from these attacks for days. In such cases there are a dilated ventricle and an enlargement of the liver and spleen.

The **prognosis** of chronic valvular disease in childhood depends very much on the type of disease. If the heart is only slightly affected and the patient not a rheumatic subject, the outlook is good. With careful management all ill after-effects can be avoided; children thus affected may grow to adult life without suffering from any symptoms referable to the heart. If, on the other hand, they are attacked by any intercurrent disease, such as scarlet fever, the heart may again become the seat of inflammatory processes. The patients may, however, recover and continue free from symptoms for years. The rheumatic cases give the most unfavorable prognosis. These are prone to recurrent attacks of endocarditis, each attack leaving the heart in a more weakened condition than before. Most of my cases have been in children who, having had one attack of rheumatic endocarditis, suffered from the affection to a greater or lesser degree for years. Within a few years of the first attack they succumb to progressive non-compensatory cardiac disease.

Treatment.—Many cases of cardiac disease in infancy and childhood give no symptoms and need very little treatment beyond careful and judicious management. Children thus affected should have a carefully regulated dietary, and should not indulge in sports which subject the heart to strain. They should not ride the bicycle, but may, however, indulge in many of the amusements of children, such as skating, roller skating, swimming to a moderate degree, and horseback exercise. They should be under constant observation, and when attacked by any acute infection however slight should be put to bed, and kept quiet until long after convalescence. In these cases an antirheumatic course is pursued even although the illness be only a mild attack of influenza or tonsillitis. It is well to give the salicylates in small doses for several days and to keep the bowels open with some alkaline cathartic. With children who suffer from rheumatism, the nature of the primary disease should not be forgotten. They should have constant antirheumatic treatment even when the cardiac disease is at a standstill. Heiman has recently shown the beneficial effects of the intermittent administration of salicylates in these cases. I have carried out that method for some time. Alkaline baths of the Nauheim form are of great utility in alleviating the subacute rheumatic pains from which these subjects suffer, and this treatment also tends to keep the rheumatic tendency in abeyance. Such children should be kept under constant observation. The temperature should be taken twice daily. Any rise of temperature should be regarded as a threatening sign and the patients put to bed for perfect rest until the crisis has passed. In cases in which there is marked dilatation or pericardial involvement, any exacerbation of symptoms is a signal for immediate rest in bed. Slight œdema of the surface and swelling of the liver and spleen will subside if treated with perfect rest, a light assimilable diet (milk), and mild alkaline catharsis. It is not always necessary to use digitalis. If given at all, it is best administered in the form of the infusion. I am accustomed to use this drug for a period of two or three days, after which I discontinue it. There is no doubt that its action continues after the administration is stopped. Convallaria in the form of the fluid extract is at times one of the most useful remedies in cases in which digitalis has failed to give relief. If there is great dyspnœa or orthopnœa, codeine in moderate doses should be used. Young children do not bear morphine well. It certainly should not be used hypodermatically. In aortic disease in older children, nitroglycerin in doses of grain $\frac{1}{100}$ (0.0006) relieves the angina. I administer morphine only to older children, and then only when the nocturnal attacks of angina are very severe. I have not found strychnine very useful in the chronic forms of cardiac disease. Caffeine in moderate dosage seems more useful in correcting the irregularity of the pulse or bradycardia seen in some of these cases. In combination with digitalis it gives excellent results. If ascites appears, the patient should be promptly tapped to relieve the circulation and the abdomen supported by a binder. If there is a pleuritic effusion at the same time, it should not be disturbed. With relief of the abdominal distention, the pleuritic effusion often disappears.

Cardiac Murmurs.

Cardiac murmurs which are the result of disease or insufficiency of the valves of the heart have the same general character as those in adults, the following being the chief points of difference:

a. Cardiac disease of a very serious character may exist (as in congenital cyanosis) without any murmur.

b. Cardiac murmurs are as a rule louder in children than in adults. The loudness is therefore no guide as to the seriousness of the affection.

c. Cardiac murmurs in children are sometimes heard loudly conducted over the whole chest; diagnosis of disease of a particular valve must be based on the greatest intensity of the murmur at that point.

d. Hæmic and dynamic murmurs in children under four years of age are not so common as is supposed. There should be no hesitation in making the diagnosis of organic affections in systolic, basic, or apex murmurs if there are distinct conduction or signs of dilatation or hypertrophy. This is especially to be remembered in chorea, extreme anæmia, and in febrile affections where rapidity in time and rhythm (galloprhythms) causes adventitious sounds.

e. The conduction of the aortic murmurs into the arteria femoralis occurs in occasional cases in children. Pulsation of the liver or spleen, as found in aortic disease of adults, is not present in children (Steffen).

Accidental Cardic Murmurs in Infancy and Childhood.

Accidental murmurs are divided into those heard over the heart, in the arteries, and in the veins. The study of the accidental murmur of the heart in infancy and childhood has been much neglected. West and Hochsinger give the most valuable data. The principal points of difference between the murmurs in infants and children and those in the adult are as follows:

Cardiac Murmurs.—*Anæmia.*—The severest forms of anæmia sometimes fail to give hæmic murmurs. Not one of 200 cases under four years of age examined by Hochsinger gave anæmic murmurs. After the fourth year and up to the seventh year of life the frequency of the anæmic and hæmic murmurs increases. I have in very exceptional pernicious anæmias found a mild blowing basic murmur. One such case occurred in a child under four years.

Fevers.—The hæmic murmurs so common in the febrile affections of adult life are rarely heard even in severe febrile affections with anæmia, in patients under the age of three years. I have heard them in children under three years of age, with severe typhoid fever. They are common in typhoid fever in older children.

Characteristics of Anæmia Murmurs.—These never occur with signs of cardiac dilatation or hypertrophy. They are not conducted into the arteries. They never entirely take the place of the valvular cardiac sounds, but accompany them. They are soft blowing murmurs, heard at times most loudly at the pulmonary valve, sometimes heard over the base and whole præcordium, and faintly heard at the apex. They are never heard at the aortic or tricuspid valves, or behind. They are inconstant, disappearing for a time and again appearing at the various points of the chest.

Accidental Arterial Murmurs.—The theory held by some observers, that pressure of the stethoscope on the arteries of the neck may cause a murmur, should be entertained with caution. Correct stethoscopy will hardly lead to such an error. A murmur in the large arteries of the neck is conducted from the heart and is invariably organic in origin. I have heard aortic murmurs conducted in the femoral artery.

Venous Hum.—Although cardiac accidental murmurs due to anæmia are rarely heard in children, the venous hum due to the same cause is frequently heard. In young infants and children it is present in the veins of the neck, is quite loud, and is heard at either side of the upper part of the sternum. If there is anæmia due to valvular cardiac disease, the venous hum is heard in the arteries of the neck, with the organic murmur.

Myocarditis.

Myocarditis is very frequent in infancy and childhood. Most of the knowledge of this condition has been obtained from a study of the disease in young subjects. This is due to the fact that in early life the heart is especially exposed to the deleterious action of the toxins of the infectious diseases. Myocarditis is a degeneration or inflammation of the muscular substance of the heart, secondary to the action of poisons (phosphorus) to the toxins of bacteria (as in the exanthemata, typhoid fever, diphtheria, pertussis, sepsis, osteomyelitis), or to the changes consequent upon disease of the pericardium, or endocardium, of rheumatic or infectious origin.

Morbid Anatomy.—If there is degeneration of the myocardium, only the muscular fibre may be the seat of fatty changes. There is an increase of fat drops in the muscular tissue of the heart. In advanced conditions, the fatty changes are apparent to the naked eye as a yellowish discoloration beneath the endocardium. In other cases, there is a granular or hyaline degeneration of the muscle fibre or a vacuole formation. The cell protoplasm becomes cloudy, hyaline, loses its striation, and disintegrates or is replaced by drops of fluid. This occurs in diphtheria, typhoid fever, pneumonia, chronic congestion, and in toxæmia of various kinds. Thrombi may form in hearts which are the seat of advanced degeneration. In toxæmia and the infectious diseases, there is inflammation of the myocardium. There is an invasion of the muscle tissue by bacteria from the endocardium (staphylococci, streptococci, and pneumococci). In such cases, there are also grayish or yellowish discoloration of the muscle tissue, vacuolization, and granular and hyaline degeneration. The muscle tissue is the seat of small cell infiltration or there may be abscesses of microscopic or macroscopic size. If recovery occurs these areas may cicatrize with connective tissue. Tuberculous and syphilitic inflammations of the myocardium occur, but are uncommon.

Etiology.—The degenerative or inflammatory changes may be caused by the direct action of the bacteria (Almquist), but usually the influence of the bacteria themselves is only slight, since they do not find in the myocardium a favorable soil for growth. The toxins of these bacteria produced either elsewhere in the economy and circulating in the blood, or in the heart muscle itself, are chiefly instrumental in causing the degenerative changes (Welch, Flexner, Schamshin). Fever, as such, has only a slight influence in causing myocarditis (Werhofsky).

The **symptoms** of myocarditis can best be understood by studying the heart in the various infectious diseases. In diphtheria, myocarditis may be suspected if there occur sudden syncope, faintness, chilly sensations, vertigo, and vomiting. The patients complain of præcordial weakness ; there are all the symptoms of collapse and a flickering, irregular pulse. These phenomena may appear at intervals throughout the disease and persist far into convalescence. In this disease there is during convalescence an irregularity of the heart apparent in the rhythm and force. There will be two or three beats and then an interval, followed by two or three beats. The pulse at the wrist may be of varying compressibility. In these cases there may be no other manifestation of the effect of the poison of the disease on the heart-muscle and ganglia. There is no pain, no vomiting, no præcordial distress, yet for days the heart-action will remain irregular and cause great uneasiness to the physician. Such cases may make a good recovery. In some exceptional cases, however, these symptoms precede more serious disturbances of a severe and even fatal character. The forms of marked cardiac irregularity are especially disquieting if observed during or after diphtheria, even of a mild type. In these cases the physician is ill at ease on account of the well-known occurrence of sudden death in this disease. I have seen irregularity persist in these cases for weeks, to disappear finally ; and yet during all this time the physician can give no positive assurance that the case may not result fatally. Simple irregularity, as a rule, without signs of true muscular weakness of the heart, such as swelling of the liver or dilatation of the ventricle, retrogrades to the normal.

In acute forms of pneumonia in which the toxæmia is very great, infants may, even at the outset, exhibit cardiac weakness. There are slight cyanosis of the lips and abnormal pallor of the face and general surface. The heart action is more rapid than in other cases of pneumonia in which the lung lesion is quite as extensive. At the crisis, the action of the poison on the heart is evinced by an irregularity or arhythmia of the pulse. The pulse may be extremely slow (bradycardia). In septic conditions there will, late in the disease, be galloprhythm, distortion of the pulse-respiration ratio, cyanosis, and extreme præcordial distress. Henoch, Osler, and I have shown that there may be degenerative changes in pertussis. These are clinically apparent in cases which have extended over a long period. A constant dyspnœa, an abnormally high pulse-rate, drowsiness, disinclination to exertion, and slight œdema of the face and other parts of the body are present. In rare cases physical examination reveals a slight dilatation of the right ventricle. In other cases there is at the apex a faint systolic murmur of purely muscular origin. In adherent pericardium, the advance of the process into the myocardium is indicated by the symptoms above detailed.

The myocarditis of chronic valvular disease is a progressive process. It manifests itself by the signs of lack of compensation described in the section on Chronic Valvular Disease. The varying pulse, the dyspnœa, the enlargement of the liver and spleen, and transudates into the serous cavities, all indicate this form of progressive weakness of the cardiac muscle.

Diagnosis.—Although the diagnosis cannot in all cases be made with absolute certainty, the presence of the condition may be suspected if the following sets of symptoms appear at regular intervals in the course of the disease—attacks of palpitation and faintness, pallor, cardiac irregularity, galloprhythm and weakness of the apex beat and of the first muscular sound of the heart, with intensification of the second pulmonic sound.

The **treatment** should support the heart and lessen its work, and should also be directed toward the management of the primary condition. In all of these cases, prolonged rest for the heart, continued long after convalescence, is of primary importance. It should not be forgotten that even in a degenerated organ there is healthy tissue on which the drugs and treatment act. These healthy foci should be sustained, and not exhausted by the action of powerful drugs given in large doses. Degeneration cannot be cured by drugs; nature must heal the diseased areas.

Cardiac irregularity pure and simple, with a pulse of moderate slowness, is best treated by means of strychnia and caffeine. To a child of three or four years of age, strychnia, $\frac{1}{100}$ grain, is given with or without a grain of caffeine every three hours. Warmth is applied to the heart, and if the extremities are cold, warm bottles are applied also. Camphor is a very excellent remedy, but can only be used for a short length of time, for it is badly borne by the stomach, and in such cases must be used hypodermically. Oil of camphor, 30 minims, may be given to a child three years of age. Severe cases accompanied by a galloprhythm are treated with digitalis. This drug is an excellent remedy in these cases, but must be used cautiously, in small doses. To a child of three or four years of age, ♏ij of the tincture of digitalis given every three hours is sufficient. If restlessness or vomiting appear, morphia is our only safeguard, but should be used cautiously. Enough only is given to quiet the patient. One or two minims of Magendie's solution is given to a child three to five years of age, by the mouth.

Hypertrophy and Dilatation of the Heart.

Cardiac hypertrophy and dilatation, combined or singly, and without any valvular lesion, occur in isolated cases in childhood. The condition is rare before the fifth year. A number of cases occurring between the fifth and the tenth year have been reported. If hypertrophy alone is present, it may affect the left ventricle only, or both ventricles. Dilatation usually affects first the right ventricle and then the left. The condition develops as a result of toxæmic influences, in the acute infectious diseases, such as scarlet fever, pneumonia, diphtheria, and typhoid fever.

Hypertrophy with or without dilatation is one of the sequelæ of acute or chronic nephritis. The nephritis complicating scarlet fever is frequently the cause of cardiac hypertrophy with or without dilatation. Atheromatous conditions of the arterial system with diminution of the calibre of the aorta may cause hypertrophy with or without dilatation. Acute dilatation as a result of heart strain is unknown in children.

The **symptoms** are not characteristic. In the absence of all other heart lesions, the diagnosis of cardiac hypertrophy or dilatation is made from the physical signs. These do not differ from those found in the adult subject. The rational symptoms also resemble those of the adult. In dilatation of the heart, there are the irregular heart action, the dyspnœa or orthopnœa, the pallor of the surface, cyanosis, and in the later stages swelling of the liver and spleen. Transudates in the pleural and abdominal cavities are apt to occur toward the close. Sudden death has occurred in some cases of dilatation of the acute variety. In hypertrophy, the symptoms closely resemble those just detailed. At the bedside, the diagnosis of hypertrophy, of dilatation, or of both, must of necessity rest on the physical signs.

The **treatment** varies with the nature of the primary disease (nephritis or toxæmia) present. The nephritis should be treated and the heart will take care of itself. If there is an infectious disease, such as typhoid fever, diphtheria, or scarlet fever, both the heart and the primary affection should be treated.

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SECTION VIII.

CONSTITUTIONAL DISEASES.

I. RACHITIS.

(*Rickets.*)

RACHITIS is a disease of nutrition causing well-marked changes in the structure and form of the growing bones. It is peculiar to infancy and childhood, and does not occur after the skeleton is formed.

Etiology.—There are two forms of rachitis, the congenital or fœtal and the post-natal.

The occurrence of congenital, fœtal, or intra-uterine rachitis is still a subject of much difference of opinion. According to some authorities (Kassowitz), 80 per cent. of the infants of the Vienna Maternity Hospital show evidences of rachitis. Epstein at one time demonstrated the great frequency of rachitic deformity at the costochondral junction of the ribs, in the infants of the Maternity Hospital in Prague.

Congenital Rachitis.—There can be no doubt of the existence of such a condition as rachitis *in utero,* or congenital rachitis. In these cases the infant at birth has craniotabes, or, if closely examined, the rosary and other marks of the true rachitic process on the long bones may easily be made out. We must not confound such cases with what has been called fœtal rickets. The latter term, as will be seen, has been practically abandoned, and was at one time applied to cases of chondrodystrophia. It is not at all a rachitic process, and has nothing in common with rachitis. Virchow insists that fœtal rachitis in the true sense is rare, and that an anomaly in the development of the primordial cartilage has been mistaken for rachitis, with which it has nothing in common.

Hemorrhagic rachitis is a term applied by some authors to Barlow's disease or infantile scurvy. Rachitis is for the most part post-natal, and its onset occurs most frequently during the first year of life. It is rare after the third year. The sexes are equally subject to the disease. A moist climate favors it. It is very common in

Germany and Austria, and is rarely met in southern Asia or Central America. Fischl insists that it is peculiar to some races of people, and Snow, of Buffalo, has shown that Italians living in America, are peculiarly subject to it. It is most common among civilized communities, in which infants, especially those of large cities, are fed upon substitutes for breast milk. On the other hand, breast-fed infants may develop rachitis, but in such cases investigation of the milk by Pfeiffer and others has not resulted in the discovery of any peculiarity of the milk which might be looked upon as a causative factor. Rachitis develops in infants who have been weaned from the breast early and fed on artificial foods or sterilized milk. The early introduction of meats and solid food into the dietary of the infant has been cited as an etiological factor.

That syphilis is a direct causative agent in rachitis (Parrot) can no longer be accepted. Heredity does not seem to exert any influence. There are many theories as to the active and immediate causes. The principal theories are those which presuppose the lack of some element, such as phosphates or lime salts, in the food, and those that trace the processes of rachitis to a disturbance of nutritive functions caused by an increase of certain acids (lactic) in the stomach, a diminution of others (hydrochloric) and resulting intestinal functional irregularities (Monti, Zander). The intestinal disturbances cause the elimination of certain salts from food, hence the blood fails to receive what is necessary for the structure and formation of the bones.

Morbid Anatomy.—Rachitis is anatomically characterized by processes which cause an increased resorption of bone, deficient calcification of cartilage, and the formation of a characteristic tissue—a deficiently calcified bone, the so-called osteoid tissue (Ziegler, Kassowitz, Schmorl). The increased resorption consists in an augmentation of the number of areas of lacunar absorption. In marked rachitis the greater part of the bony skeleton is lost. The cortical area of the long and of the short bones becomes osteoporous. A large part of the lamellæ of the cancellous bone is absorbed and disappears. In the flat bones the arrangement of outer and inner table separated by the intervening diploë is lost. The bone tissue is reduced to a few lamellæ. At the zones of periosteal and medullary ossification, the lamellæ are replaced by osteoid tissue. This tissue is a new formation devoid of lime salts.

The marrow of the osteoid tissue formed from the periosteum or medullary canal consists of a reticulum of striated connective tissue rich in bloodvessels and enclosing free round cells. Beneath the periosteum of the cranial and long bones there is formed, because of these changes, a spongy vascular tissue which is resistant to pressure and may be cut with a knife. While the rachitic process lasts, no lime salts appear in the lamellæ of osteoid tissue, but as soon as the disease has spent itself those salts appear in the centre of the lamellæ. Complete recovery results in calcification of these lamellæ, which being proliferated leave the bone hardened and very much thickened. The pathological change in the endochondral ossification consists in an entire absence of a calcification zone. In severe rachitis, all signs of the deposit of lime salts are absent. There is a widening of the zone of proliferation of cartilage cells, and also of the columns of hypertrophoid cartilage cells. There is lastly an irregular formation of vascular marrow-spaces, which grow here and there into the cartilage from the bone. Thus at the junction of cartilage and bone, there is in the long bones no distinct line of ossification. The red marrow-spaces extend for varying distances into the cartilage.

The abundant growth of bloodvessels extending from the perichondrium into the cartilage is accompanied by the substitution of osteoid tissue and marrow-spaces for the cartilage proper, as in periosteal and medullary ossifications. In rachitis the cartilage is never completely absorbed by osteoid tissue. Thus, on section, the bone shows, nearest the cartilage, the zone of proliferating cartilage-cells with hypertrophied cells in columns; next to this is the zone of osteoid tissue in lamellæ in which few lime salts are deposited. Nearer the bone are lamellæ of osteoid tissue, in the centre of which fully formed bone is deposited. The lamellæ of osteoid tissue differ from those of normal bone in being much thicker and more abundant. The osteoid tissue is very resilient and easily bent, hence this property of rachitic bones. The process leaves the bones much thickened, especially at the epiphyseal extremities. The deformities of the chest, extremities, pelvis, and spine can thus be traced to the tendency of the rachitic bone to bend on pressure and traction. The effects of the process on the shape of the cranium and the delay in the formation of the teeth may thus be easily accounted for.

Among other gross lesions connected with the clinical picture of rachitis is enlargement of the spleen. The organ may be very large and easily palpated below the border of the ribs. Sasuchin found that of 66 cases of rachitis, the spleen was enlarged in 12 to 15 per cent. The changes in the organ consisted in thickening of the capsule and proliferation of the connective tissue of the organ, arteritis, thickening of the walls of the arteries, atrophy and obliteration of the Malpighian bodies, and anæmia of the organ. This important blood-distributing organ is thus compromised. The spleen may be increased to two and a half times its normal size. The liver may also be apparently enlarged. During life the enlargement of the liver may be more apparant than real. The chest, if narrow and deformed, may cause downward displacement and rotation of that organ. In rachitic infants the lymph-nodes are more apparent on palpation than is normal. They, however, are never increased to the size attained in tuberculosis, syphilis, or eruptions of the skin, such as those of the exanthemata. The blood may show the changes of extreme simple anæmia—an increase in the nucleated red blood-cells and other signs.

Brain.—Slight or marked hydrocephalus is frequently found in rachitis. The relation between the two conditions is not clear. If the infant dies of an intercurrent disease, changes of a chronic catarrhal character may be found in the gut and signs of bronchitis or persistent bronchopneumonia in the lungs. These conditions follow the changes in nutrition which cause the rachitic processes elsewhere.

Symptoms.—The most marked and general symptoms of rachitis are changes in the bony skeleton.

The Head.—The shape of the rachitic head is very characteristic. The frontal bone bulges, giving the infant a very prominent forehead. The parietal bones have a flare, caused by the formation of bosses at the centres of ossification. The whole head has a cuboidal shape, which, with the proportionately small face, gives the characteristic appearance. The disturbances in bone formation cause the appearance of soft spots, especially in the vicinity of the lambdoidal suture. These (craniotabes) may be membranous in structure. They rarely appear on the frontal bones in the vicinity of the coronary suture. The spots of craniotabes appear in infants who develop rachitis before the sixth month (Monti), rarely after this period. They take four or five weeks to develop fully. In developed rachitis the occiput is flat and devoid of hair (Plate XIX.). The anterior fontanelle,[1] which normally closes between the fifteenth and the eighteenth month, remains open for a long time, in some cases until the third or fourth year, or even to the sixth. The sutures are also slow in closing. The coronary sutures may remain open for two, and the longitudinal suture for three years. The lambdoidal suture does not in some cases close until the eighteenth month.

If the thorax is affected by rachitis, the circumference of the head will exceed that of the chest. The lower jaw has an angular deformity, described by Fleischmann. This consists in a bending of the body of the jaw at the situation of the canine teeth. The body of the jaw is also rotated internally on its horizontal axis. If rachitis begins before the sixth month, dentition is delayed for periods varying up to a year and a half. I have a record of a case in which the first tooth appeared at the twenty-fourth month. If rachitis develops after appearance of the first teeth, the succeeding ones appear

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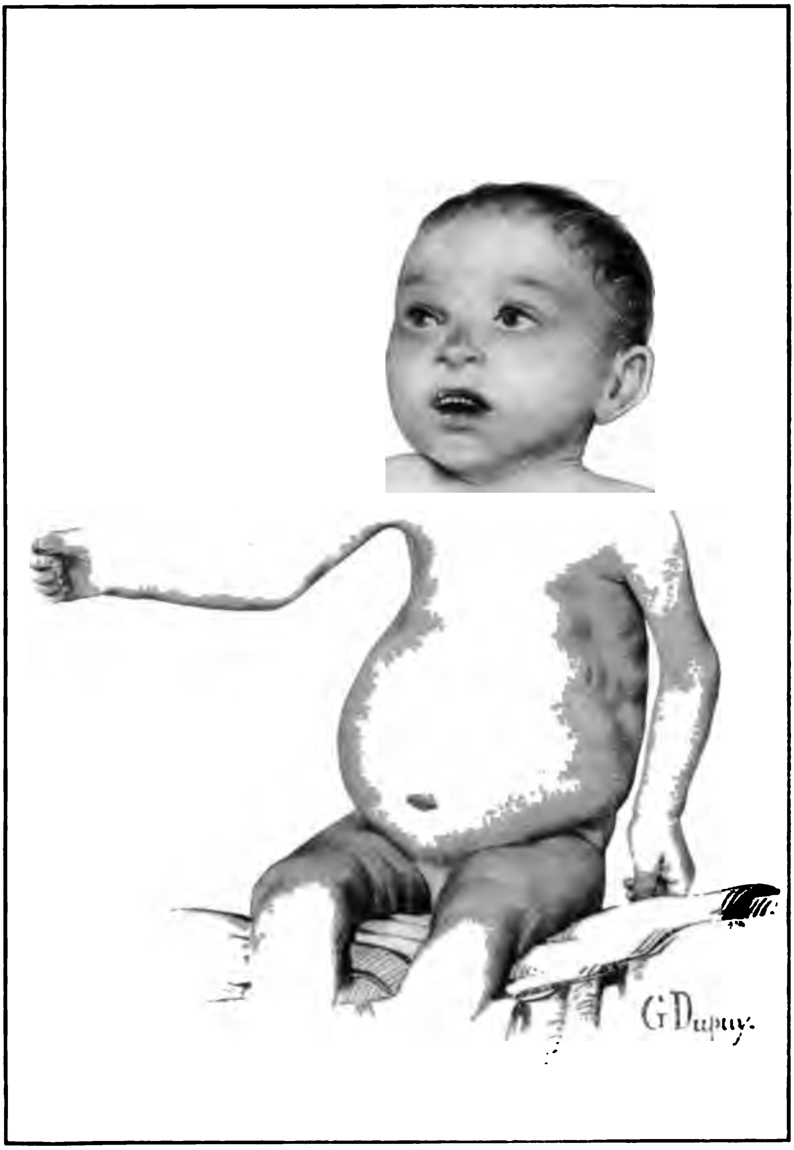
[1] While the lateral and posterior fontanelles close during the first months of infancy, the anterior fontanelle increases in its longitudinal and transverse diameter with the growth of the cranium up to the twelfth month. The growth of the anterior fontanelle was first observed by Elsässer. Alhough denied by Kassowitz it has been recently proved by Rhode that the contention of Elsässer is correct.

PLATE XIX.

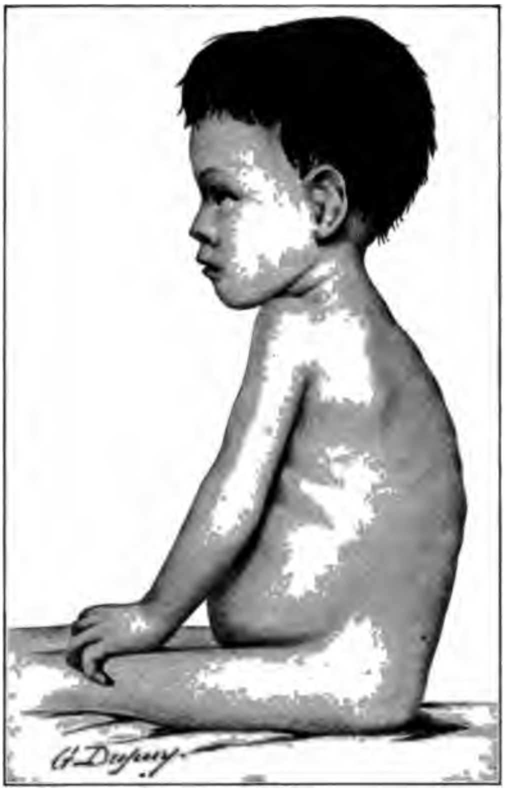


Rachitis. Showing the cuboidal shape of the head, the thoracic deformity, the beaded ribs, the protuberant abdomen, and the enlarged lower end of the radius.

later than is normal. The structure of the teeth suffers. They show erosions, are easily broken, and become carious quickly. This is due to imperfect formation of enamel or dentine. Some time after their eruption, the incisors show a well-marked incurvation at the free border, which is due to erosion or breaking of the tooth.

The **thorax** shows very characteristic deformities. Rachitis of the thorax in most cases develops in the second half year, and may continue into the third year. The first marked sign is the appearance of the so-called rib rosary. This is a thickening of the costochondral junction of the rib, in which the rachitic processes above described are very active. Deformity of the thorax follows in course of time. The thorax becomes prominent at the sternum and flattened in the midaxillary region from the axilla to the free border of the ribs. There is a distinct incurvation of the thorax above, and a flaring below. The thorax is much narrowed at the clavicles, with a flaring outward of the lower ribs. Respiration, especially inspira-

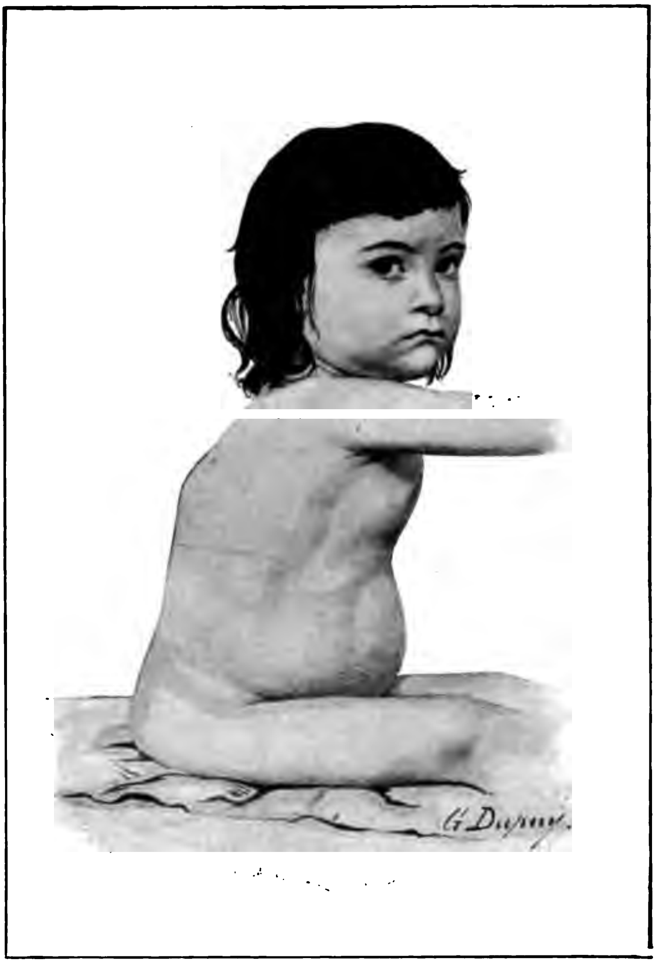
FIG. 145.



Rachitic deformity of the spine. Uniform curvature backward.

tion, is much interfered with. The sides of the thorax are drawn inward at the diaphragm at each inspiration. In an attack of severe bronchitis or bronchopneumonia, the drawing inward of the sides of the chest becomes still more marked. In some cases the sternum alone is affected. There is a sinking of the sternum, with resulting chest deformity. Some forms of rachitis affect only the ribs or part of the thorax. While the rachitic process is in progress, the chest circumference does not increase; it begins to do so when the disease has run its course in the thorax.

FIG. 146.



Angular deformity of the spine, due to Pott's disease, as distinguished from the deformity due to rachitis.

Pain.—When the infant is raised from the chair or crib, it cries. This is the result of the painful nature of the rachitic process in the bones. Forcible percussion of the chest will cause pain. On account of the deformity of the chest and the consequent interference with its physiological functions, the lung is prone to contract infections, such as bronchitis and bronchopneumonia. Atelectasis is also a common complication. The clavicle becomes bent and fractures on the slightest traumatism. At the termination of the rachitic process, the clavicle and scapulæ are much thickened. Virchow has shown that the scapula becomes the seat of an angular deformity.

Spine.—On account of the relaxation of the ligaments of the bodies of the vertebræ and of the rachitic processes in the bodies of the bones themselves, there is in most rachitic infants a bending backward of the dorsolumbar spine (Fig. 145). The curvature is very marked when the infants are held in the arms. It differs from deformity due to Pott's disease in that it is not angular, and in that the spine can be straightened and even curved forward with ease (Fig. 146).

Lateral curvatures of the spine are also found. If the spinal deformities occur early in infancy, they disappear as the rachitis heals and the ligaments and muscles regain a normal tonicity. On the other hand, should the rachitic process attack the spine late in the third or fourth year, the deformities are perpetuated. This is especially the case if the pelvis is also affected at that time (Monti).

The **pelvic deformities** which result from rachitis are chiefly flattening of the pelvis, and the pseudo-osteomalachic pelvis.

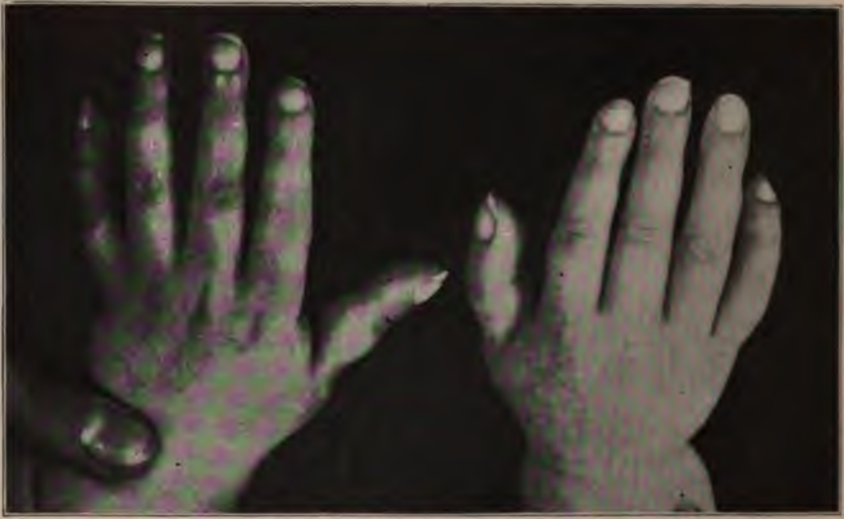
Upper Extremities.—The epiphyses are much swollen and, in rare cases, painful. The wrist is flat and much broadened. If the rachitis is elsewhere not marked, the physician should be careful not to mistake a normal enlargement in this situation for rachitis. In exceptional cases, the elbow and shoulder-joint show similar changes.

On account of the traction of the flexors and pronators, the forearm may be incurvated and the bones twisted on their longitudinal axes. The result is a more or less fixed position of pronation in the forearm. The arm is rarely curved in this manner, but it may, like the clavicle, be fractured after slight traumatism. As a result of rachitis and deformity, the growth of the bone in length is much interfered with.

The phalanges are sometimes the seat of the rachitic processes. In some severe cases I found all the phalanges thickened in the diaphyses. These cases bear a very close resemblance to dactylitis syphilitica, especially as there is pain on pressure (Fig. 147).

The deformities of the **lower** extremities are more marked than those of the upper ones. On account of the pain experienced, the infants refuse to stand; they will draw the extremities up underneath the abdomen, if any effort is made to make them do so. In other cases, when attempts are made to stand, the weight of the body and the muscular traction (Kassowitz) cause deformity. The femur, tibiæ, and fibulæ curve outward, giving the so-called "bow-leg" deformity (Plate XX.). This may in extreme cases result in a deformity of the heads of the bones entering into the formation of the knee-joint. The ankle-joint may suffer a varus deformity. The femur and tibiæ may curve inward, and a knock-knee deformity result. In all cases, there is relaxation of the ligamentous joint-structure. The tibia sometimes becomes much thickened and curves anteriorly, giving the so-called "sabre deformity." It may be twisted on its longitudinal axis. I have seen severe rachitis of the femur and tibia result in multiple fractures.

FIG. 147.



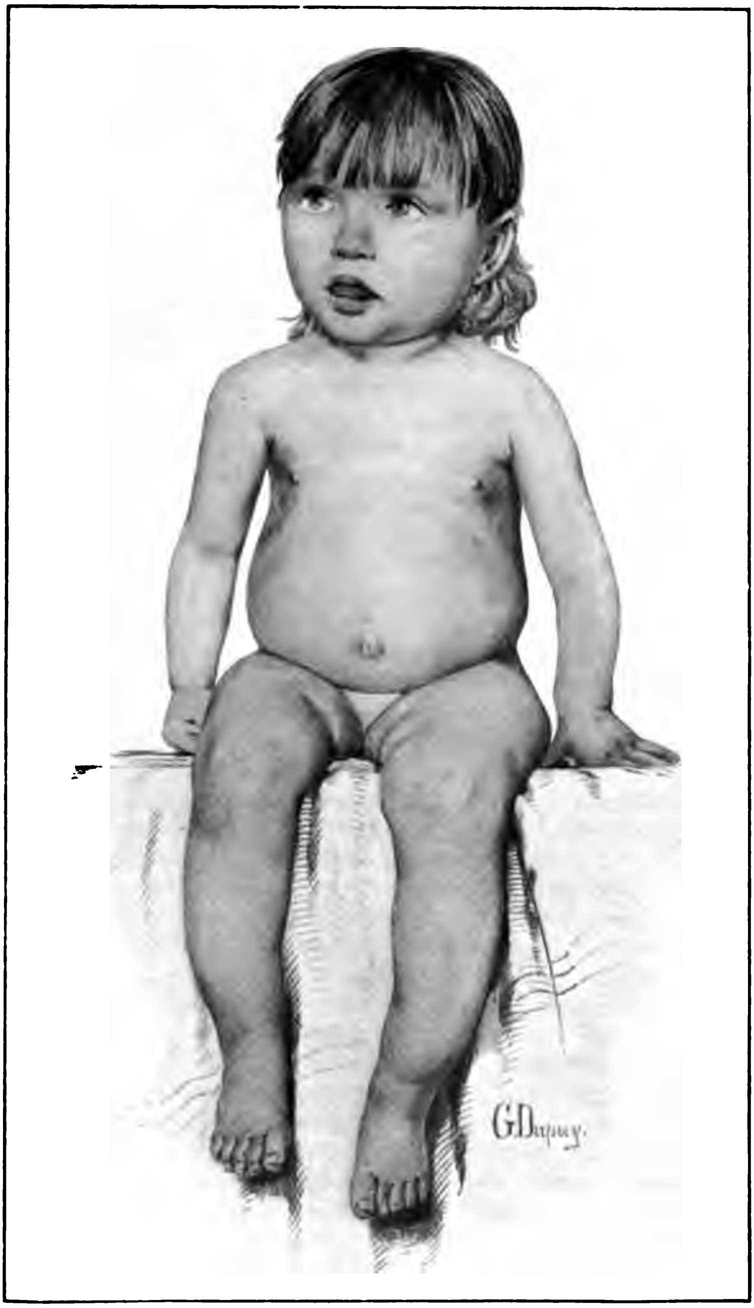
Rachitic hands, showing bowing and thickening phalanges of fingers (author's case).

The deformity at the hip-joint, which later in life follows changes in the angle made by the neck of the bone with the shaft of the femur (coxa vara), is believed to be due (Whitman) to rachitis. The children are late in walking. The musculature is weakened through disuse.

When the children assume the sitting posture, they cross the lower extremities in tailor fashion. In the majority of cases of rachitis, the abdomen is protuberant. As a result of the defective nutrition, the musculature of the gut is weakened in the same manner as that of the extremities. Tympanitic distention is the rule.

Intestinal disturbances are common in rachitis, but are not a result of the process. Henoch shows that rachitis may be present with an apparently normally functionating intestine.

PLATE XX.



Rachitis. Showing the deformity of the thorax and marked bowing of the tibiæ.

The **spleen** is enlarged in many cases of rachitis, but retrogrades to the normal size after the disease has run its course.

The **blood** shows the changes found in ordinary mild or severe simple anæmia.

The **liver** may be slightly enlarged.

Anæmia of the skin and mucous membranes is frequently found. It may be so extreme as to cause the skin to have a yellowish waxy hue. Rachitic children perspire freely at night, especially about the head. Unless the skin is kept scrupulously clean, sudamina, furuncles, and eczema of all kinds will result.

Nervous System.—There is no doubt that certain nervous affections, such as tetany, laryngismus stridulus, attacks of inspiratory apnœa, spasmus nutans, and the so-called barn-yard crowing or congenital stridor of the larynx (described by Thomson), occur most frequently in subjects of rachitis. Some authors (Kassowitz, Jacobi, Escherich) trace a distinct etiological connection between these conditions of instability of the nervous system and rachitis.

Hydrocephalus occurs in rachitic subjects. In cases of severe rachitis, an appearance of mild hydrocephalus is given to the face by a downward depression of the eyeball. The sclera of the eyes is thus slightly exposed. The appearance seems to be caused by a depression of the orbital plates of the frontal bone by the overlying frontal lobes of the cerebrum. In many cases of severe rachitis, the wide fontanelle, its tenseness, and the open coronal and temporal sutures give a picture like that of a non-progressive, mild hydrocephalus which is simply a feature of the nutritive disturbances taking place in the brain as elsewhere.

Severity of the Affection.—These symptoms are not present in all cases of rachitis. In some cases there are only very slight signs of the disease, such as a slightly cuboidal shape of the head or a scarcely appreciable bending of the ribs without any deformity. In such cases even an expert may be in doubt as to the presence of swelling of the epiphyses. In other cases an intercurrent affection, such as tetany, will cause the physician to seek for signs of rachitis, which may be so slight as to have previously escaped notice. Craniotabes is sometimes absent in marked cases. Delayed dentition is not the rule. Rachitis may be very evident in cases in which the teeth appear in their normal order.

Duration.—In such a disease as rachitis it is to be expected that the duration of the affection will vary greatly in different subjects; it may last months in some cases, in others years. The first favorable sign is the attempt of the infant or child to walk, but children with marked and progressive rachitis sometimes walk early.

Increase in weight and in the chest circumference, an improvement in symptoms, such as anæmia and intestinal disturbances, and the cessation of pulmonary complications are indications that the disease has come to a standstill.

The **diagnosis** of rachitis before the development of the physical signs in the bones of the head, chest, and extremities is scarcely possible. Monti thinks that an increase of lactic acid in the stomach contents is, if there are intestinal disturbances, strong présumptive evidence of early rachitis, but the increase of lactic acid may be temporary, and the general practitioner will find it hard to estimate. Once the bone symptoms develop, there is no difficulty. In cretinism, Mongolian idiocy, and syphilis, there are changes in the bones which very closely resemble those seen in simple rachitis. Yet in all these conditions there are other signs which will make the diagnosis clear. In syphilis, rachitis is an accompanying condition. There is no etiological connection between the two affections. In every case of tetany, spasmus nutans, laryngismus, congenital stridor of the larynx, inspiratory apnœa, or eclampsia, the physician should not fail to look for evidences of rachitis. The improvement in these conditions will often depend on the management of the rachitis.

If the infant cannot stand, the limbs may exhibit a variety of pseudoparalysis. Paralysis may be excluded by making an electrical muscle test. Although infants with rachitis will not stand, they move the lower extremities vigorously when lying down. This is not the case in the palsies; the faradic and galvanic muscle tests and the presence of the normal reflexes will fix the diagnosis. In severe cases of cranial rachitis, it is not always an easy task to exclude hydrocephalus. While marked hydrocephalus presents no difficulties, a slight hydrocephalus is not always apparent. In such cases the head circumference is measured once a month. An abnormal increase in the circumference, a wide tense fontanelle, and open sutures indicate hydrocephalus.

Occurrence.—West has demonstrated that rachitis in the United States is not confined to negroes and immigrants. He has shown that its greatest frequency is among the natives of Eastern Ohio.

The Blood.—Through a study of the blood in rachitis Morse has come to the conclusion that anæmia of any form may exist. It is generally an anæmia in which the number of red blood-cells is normal or nearly so. The hæmoglobin is reduced, and there is a consequent reduction in specific gravity. There is leucocytosis, especially in the cases with splenic enlargement.

Rachitis tarda is a term applied by Kassowitz and Genser to those cases which, instead of running their course in two or at most three years, continue in the active stage for eight, ten, or even twelve years. Kassowitz and his pupils record cases of florid rachitis at the tenth and twelfth year. I have seen a case of florid rachitis in a female child eight years of age. She had all the signs of rachitis of the head, thorax, and arms. The lower extremities were permanently crossed in tailor fashion. The bones were painful, and those of the lower extremities were the seat of multiple fractures. The teeth were decayed. In Genser's case the milk teeth having decayed and fallen out, the permanent ones failed to appear.

Prognosis.—If rachitis is not complicated by any intercurrent affection, the prognosis, even in the severe forms, is generally good so far as life is concerned. On the other hand, an intercurrent affection, such as pertussis or bronchopneumonia, is likely to run a severe course and prove fatal in a rachitic subject. If the rachitic process is complicated by nervous disorders, it is frequently fatal. Sudden death in eclampsia, tetany, or laryngismus is not uncommon.

The prognosis as to deformity will depend on the severity of the affection. Subsequent treatment will not always correct deformity of the pelvis and long bones. The conditions often remain permanent. Fortunately rachitis in this country is not among the native born of so severe a type as in Germany, Austria, and Switzerland. If marked hydrocephalus is a complicating condition, the prognosis is bad.

The **treatment** of rachitis differs greatly in different countries, but there are certain fixed principles upon which all methods are based. Prophylaxis is an important element in all methods. An infant at the breast should not be weaned too soon if the breast milk is sufficient in quantity and the infant is increasing in weight. Weaning should not be attempted until the ninth month. If it is done in the fall or winter, the milk should be obtained as soon as possible after the time of milking. There is no need of sterilizing the milk if it has been collected with care. It is at most pasteurized. Cows' milk should be diluted so that the albuminoid elements may be reduced. Articles of diet rich in albumins, such as eggs, should not be given early, nor should the infant be permitted to eat meat in any form, potatoes or vegetables. The early use of these articles of diet favors the development of rachitis. When the breast milk is insufficient, it should be supplemented by the requisite number of artificial feedings. Rachitic infants do better on two breast-feedings a day with several artificial feedings, than on artificial feeding alone. Cows' milk is the substitute for the breast. It should be properly prepared. Many severe forms of rachitis can be traced to the use of infant foods.

Artificially fed infants should, after the sixth month, be allowed a limited amount of fresh fruit juice once a day. Orange juice is best, but cannot be borne by all infants. An infant should not be allowed to become inordinately constipated. In other words, treatment is directed toward eliminating all predisposing factors to the development of the disease. Some breast-fed infants do not thrive. They develop serious disturbances of nutrition and colic, remain stationary in weight, and have irregular and green curdy movements.

In such cases, the infant should be weaned or given another wet-nurse. Damp, ill-ventilated dwellings predispose to the development of rachitis.

Bathing.—Young infants should not be bathed in water which is much below the temperature of the body. Such bathing prevents increase in weight and causes disturbances of nutrition. The temperature of the bath should be practically the same throughout infancy. An infant cannot be hardened without disturbing the metabolism. The addition of sea salt to the bath water is advised by some physicians, and brine baths are in general use. There are other kinds of baths which contain iron, but I have had no experience with them. They are not used in America.

Living at the sea-coast is believed to exert a very favorable influence upon rachitic infants and children. On the other hand, if there are affections of the chest and lungs, such as bronchitis of a chronic variety, the humid atmosphere of the coast is not likely to be beneficial, and mountain resorts are better.

Medicinal Treatment.—Cod-liver oil has long been a favorite drug in the treatment of rachitis. It should be given in the emulsion with the hypophosphites of lime and soda. An infant a year old should take half a teaspoonful three times daily. In intestinal disturbances, it should not be administered, for fear of aggravating the symptoms. The external application of the pure oil to the body can hardly be useful, since it certainly interferes with the metabolism of the skin.

IRON in the form of the hypophosphate, grain j (0.06) given four times a day, or the saccharated carbonate, grain ij (0.12) three times daily, is of great utility. The pomate of iron or the more digestible peptonates of iron and manganese are much used. The combination of thyroid extract and iron has, in some cases of extreme anæmia with enlarged spleen, been of great utility. I have used this combination only in cases where there was extreme anæmia with rachitis:

Thyroid ext.	gr. ½ (0.03).
Sacch. carb. iron	gr. iij (0.2).

Tabes pulv. t. i. d.

Henoch has advocated the use of thyroids in the advanced cases of rachitis. His view is opposed by other authorities (Monti). I advise the cautious use of thyroids in combination with iron in selected ambulatory cases only. Hospital cases will not do well on this therapy.

The lactophosphate of lime is advised by some authorities, but is of little value.

PHOSPHORUS.—It has been shown by Kassowitz and Wegner, and confirmed by Virchow, that in the lower animals phosphorus administered in sufficient dosage causes an increased activity in the processes at the epiphyseal ossification zone. The bone becomes more compact, but there is neither an increase of its diameter nor deformity. Kassowitz has contended that the same results are obtained in the human subject. On this question, there is wide difference of opinion. Jacobi was among the first in this country to administer phosphorus as a remedy for rachitis. He especially advises its use in cases of craniotabes. I have found that some children do well on it, while in others it causes gastric and intestinal disturbances. I have used the emulsion of lipanin, so much recommended by Kassowitz, as a vehicle for the phosphorus. Enough of the phosphorus is put into the oil to make a teaspoonful of the emulsion equal to $\frac{1}{250}$ grain (0.00024). Thompson's solution of phosphorus may also be used. Preparations of phosphorus, even those made with oil, deteriorate. Kassowitz advises the formula to be made up with recently dissolved phosphorus.

There are those who, like Henoch, Monti, and Heubner, regard the phosphorus treatment of rachitis with distrust. The treatment of rachitis with glandular extracts is still a matter of empiricism. The treatment of the convulsions of laryngismus will be discussed in the section on that condition.

Surgical Treatment.—It is not within the scope of this book to dilate on the surgical or orthopedic management of rachitic deformities. It is, however, proper to state that it is neither right nor necessary to place every infant with marked spinal curvature due to rachitis in a plaster jacket. A young infant with marked backward curvature of the spine will gradually lose this deformity as its muscles improve in tonicity, but if placed in a plaster jacket will probably develop a subacute bronchitis or pneumonia. The lung is insufficiently inflated as it is, and becomes much more so if the soft thoracic walls and abdomen are encased in a plaster cast. In such cases the sitting posture should be avoided. The infants are kept in the arms or sleep on an ordinary hair mattress and hair pillow. It is not possible to keep them in any particular posture. Massage of the spine is of questionable utility.

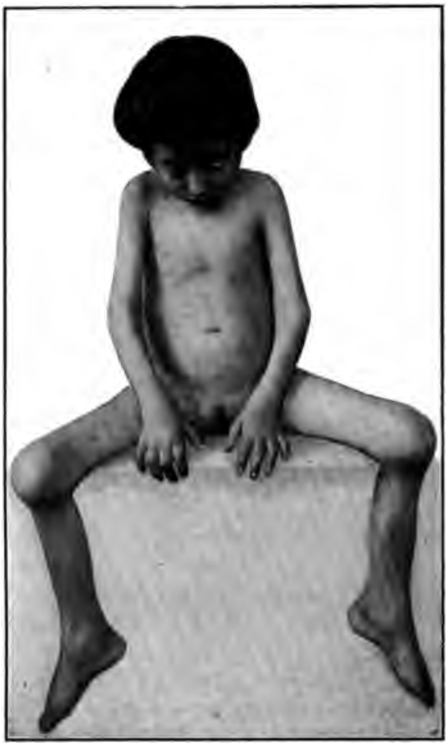
Operations for the correction of deformities of the long bones should not be carried out until the rachitic process has come to a standstill. Surgeons sometimes advise the correction of deformities in young infants by encasing the limbs in plaster while the bones are still soft.

II. RHEUMATOID ARTHRITIS.

(*Arthritis Deformans.*)

This affection should be sharply differentiated from all forms of chronic or subacute articular inflammation. Charcot and Weil have described this form of arthritis in children. The cases are not common. After the publication of my case, two others were described in the American literature, one of the descriptions being given by Manges. Cases of arthritis deformans or rheumatoid arthritis in children are referred to by Osler (4 cases) and Henoch (5 cases). The onset of the disease is either sudden after an exposure to cold and wet, or slow. In one form, after an onset of chills and fever, soreness and pain in several joints appear. The child is at first able to be about, but, as the joints become more and more affected, complete disability results. The pain in the joints becomes so marked as to interfere with sleep. After a few months the patient may be unable to walk. In some cases the enlargements and pain begin in the lower extremities and gradually involve other joints. In others the onset is slow. The joints of the upper and lower extremities gradually become painful, and after repeated attacks remain swollen and limited as to motion. The ends of the bones are enlarged and there is effusion in some joints. With

FIG. 148.



Arthritis deformans in a child seven years old. Deformity of all the joints with fixation. Child forced to assume this attitude awake and in sleep.

the progressive involvement of the joints there is atrophy of the muscles, as in the adult form of the disease. When the disease is fully developed the condition is pitiable. In my case almost every joint in the body, including those of the cervical vertebræ, was involved; the temporomaxillary articulation, the shoulder, the elbow, the small finger-joints, the hips, knees, ankles, and toes, were all affected. The patient slept in a semi-upright posture, and had to be carried from place to place. There was very limited and painful motion in all the affected joints (Fig. 148). In some cases there have been exophthalmic goitre and tachycardia (Manges); in others there also has been enlargement of the lymph-nodes, liver, and spleen.

Brabazon found that of 100 cases of this affection, only 3 per cent. occurred between the ages of five and fifteen years. Two theories have been advanced to explain this joint-affection; one, that of Charcot and Weil, is the neurotic theory, which is plausible because of the bilateral nature of the affection, the atrophy of the muscles around the joints, the changes in the skin which becomes in time tense and shining, and the enlargement of the ends of the bones which enter into the formation of the joints. The infectious theory is supported by the fact that there is in many cases a diurnal fluctuation of temperature of a degree or a fraction of a degree above the normal. The lymph-nodes are enlarged; the liver and spleen are also enlarged in some cases. The heart is not usually involved.

The **prognosis** as to life is good.

Treatment by massage, warm baths, and patient manipulation of the joints under anæsthesia, may effect slight improvement. In my case improvement was noted after a year of constant treatment. Iodide of potassium is the only drug which relieves the pain. In some cases it exerts a favorable influence upon the course of the disease.

III. CHONDRODYSTROPHIA FŒTALIS.

(*So-called Fœtal Rickets; Achondroplasia, Micromelia.*)

Definition.—This is a true dystrôphia of cartilaginous growth in the long bones, resulting in deformities which consist in a shortening of the extremities and certain changes in the bony structure of the head. Cases of this rare condition have been reported in this country by Jacobi, Smith, Herrman, and Townsend. Thomson, of Edinburgh, has described the affection as of intra-uterine origin. Although Horsley and Barlow classify these cases with sporadic cretinism, they have nothing in common either with cretinism or rachitis, and must be regarded as a distinct pathological entity. The patients are far from being idiotic or presenting any of the symptoms of myxœdema. The case published by Townsend was that of a still-born infant. Parrot and Jacobi have described infantile cases.

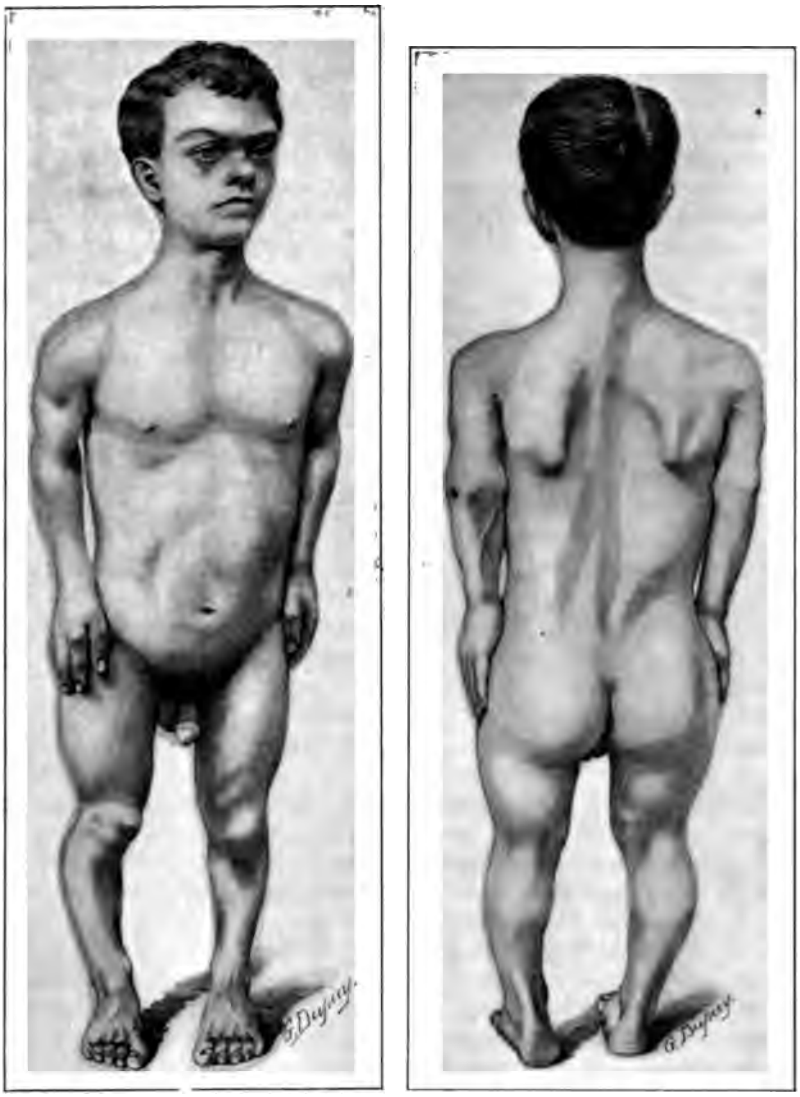
Forms.—From a pathological standpoint there are three forms of this affection : The first is that in which there is a softening of the primordial cartilage, or so-called chondromalacia fœtalis ; second, that in which there is a cessation of growth of cartilage, so-called chondrodystrophia hypoplastica ; and lastly, the form in which there is an increased but very irregular growth of the cartilaginous part of the long bones, so-called chondrodystrophia hyperplastica. In all of these forms the resulting deformities are characteristic. They are as follows :

(*a*) The skull has a peculiar form, the vertex is large. The root of the nose in one set of cases is sunken ; in another set the whole nose is flattened. In both sets of cases a peculiar expression is given to the face, which at first was mistaken for cretinoid. The form of the skull was thought by Virchow to be due to a premature synostosis of the three bones comprising the tribasilar bone ; this has since been disproved, being true of only one set of cases ; in some cases the whole tribasilar bone is cartilaginous, and in others there is no synchondrosis, nor even a marked shortening or premature synchondrosis. The changes in the skull are of the same nature as those in the long bones, viz., dystrophic.

(*b*) The long bones in the most characteristic types are shortened. The diaphysis is short and thick, so as to present little or no medullary canal ; the epiphyses are mostly cartilaginous and enlarged, and the whole bone is bent, the normal curve being exaggerated. The picture thus presented is that of a dwarf with short extremities (micromelia). There are forms of chondrodystrophia without any marked shortening of the extremities, but rather of the lower part of the trunk (Klebs, Kaufmann) (Fig. 149).

Morbid Anatomy.—There are no changes in any of the internal organs. The parts at the base of the brain, the pons, may extend above the sella turcica in an upward instead of a forward direction. This is due to the peculiar changes present at the base of the skull. The pituitary body is normal. The thyroid shows no marked changes. The flat bones are normal ; but in the bones which are formed from cartilage, the so-called endochondral ossification is disturbed. These bones, such as the sternum, patella, and costal cartilages, the tarsal and metacarpal bones, show changes. The long bones present endochondral disturbances ; there is an absence of the long lines of cartilaginous cells, and at the ossification zone there is a most irregular proliferation of cartilage-cells and ossification. It is thus that the growth of the long bones, of the innominata, and of the bones at the base of the skull are disturbed. The vertebral column may be normal, or the antero-posterior diameter of the vertebræ may be shortened. The thorax is small and flat, due to arrested development of the ribs. On section the bones present no parallel rows or cartilage-cells, no medullary spaces, no projection of medullary bloodvessels into the

FIG. 149.



Chondrodystrophy (achondroplasia). Seventeen years of age.

Anterior view, showing depressed root of nose, short extremities, peculiar articulation at the knee, and well-developed genitals.

Posterior view, showing large head, low shoulders, normal size of trunk, short scapulæ, lordosis in the lower lumbar region, and muscular legs.

(Case of Dr. Chas. Herrman.)

cartilage. There is an absence of vessels at the ossifying junction, the bone being formed mainly from the periosteum. The heads of the bones are thus chiefly made up of hyaline cartilage; the shaft of the bone of periosteal bone-formation.

From the above data of the morbid anatomy in this disease it can be seen why this condition has nothing in common with rachitis and cretinism, and should not be called fœtal rickets or "so-called" fœtal rachitis.

Symptoms.—The general picture is that of a dwarf with short extremities and a body trunk of normal length. The four extremities are affected. The arms are shorter than the forearms, the thighs than the legs. The head is large, at times simulating a hydrocephalic contour, the parietal and frontal bones are prominent, the root of the nose is broad, the bridge depressed, the tip large and the nostrils open, the features are large and heavy. The vault of the palate is high. The lumbar curve of the spine forward is much exaggerated, the sacrum thrown back, causing in the female a narrowing of the brim. The hips are large and muscular, as also the muscles of the extremities and trunk. The lower extremities are bowed and the legs are articulated at an angle with the thigh. The hands are square, massive, reduced in all proportions, the fingers of equal length, thus giving, when spread, the appearance of a trident. The intelligence is very good; in some cases the subjects may not be as bright as the normal individual.

Diagnosis.—A differential diagnosis must be made from Rachitis, Cretinism, Infantilism, and Osteogenesis imperfecta. A careful study of the symptomatology will show quite distinctly that the characteristics of each of these conditions cannot be mistaken for each other.

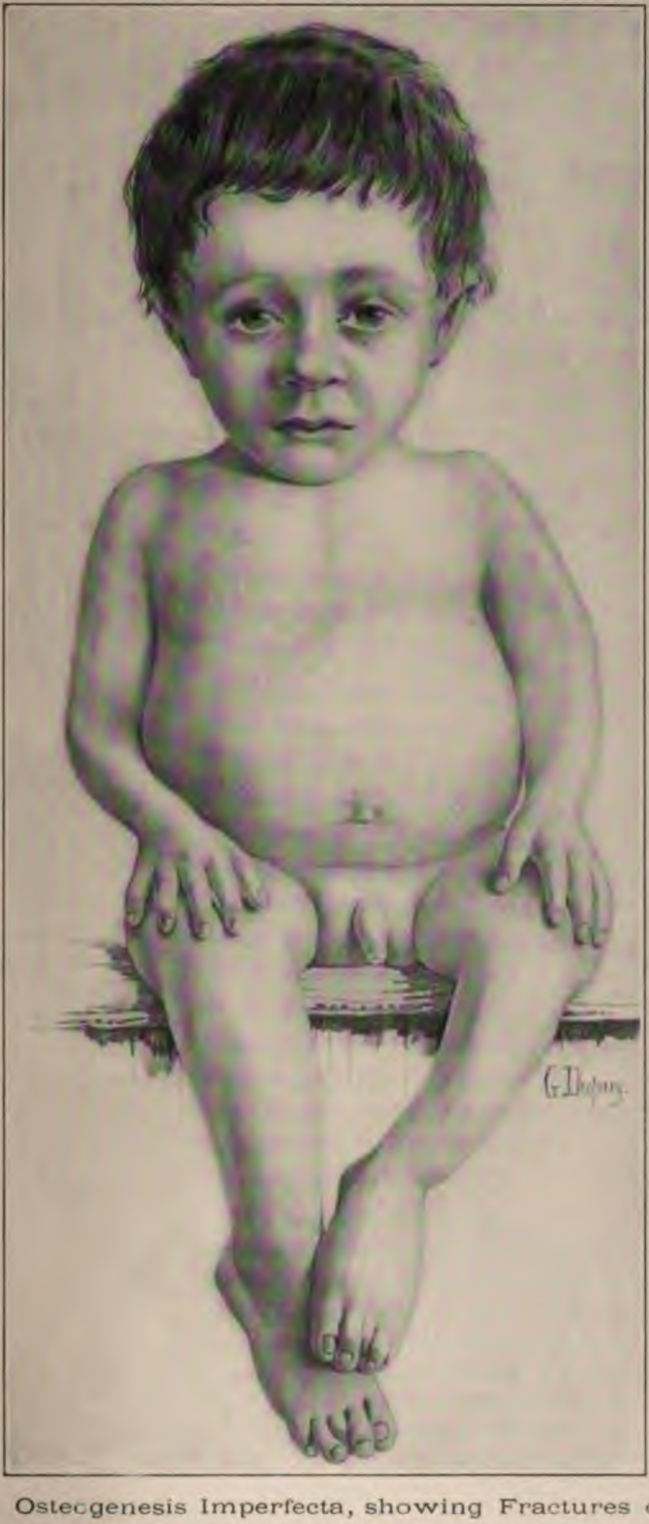
Prognosis and History.—Many of these cases die at birth, but many attain adult life and are of good intelligence, though some cases may have less than the normal intelligence. They may have children. The children of the female sex may have chondrodystrophia; though among the handsomest that I have ever seen were the offspring of a female chondrodystrophic dwarf, whose children were patients in my clinic. This dwarf had little difficulty in labor, though in some cases this difficulty may be present. Her children presented absolutely no deformities, but were brought for treatment for the slight disturbances of infancy and childhood.

OSTEOGENESIS IMPERFECTA.

(*Fragilitas Ossium Idiopathica.*)

This is a systemic disease of the bones which attacks the young fœtus and, without causing appreciable abnormalities in other organs, prevents or disturbs the normal development and calcification of osteoid tissue. The disease manifests itself by defective development of the cranial bones, with fragility of the entire osseous skele-

PLATE XXI.



Osteogenesis Imperfecta, showing Fractures of the Long Bones with Resulting Deformities. (Case of Dr. Nathan.)

ton. Cases of this nature have been reported in the fœtus or in the newborn infant—born dead or dying within a short time after birth ; recently however, cases have appeared in the literature which have lived to adolescence with all the symptoms of the affection.

Morbid Anatomy.—The examination of the bones after their removal from the body shows them to be delicate and fragile, fracturing with the slightest force. At times the periosteal bone shell is so thin that it may be crushed between the fingers with very little force. Sections of the bones show them to be porous, the trabeculæ delicate, the outer layer exceedingly thin, there being no dense bone, but a collection of small plates and trabeculæ. Calcification of the osteoid tissue is defective or entirely absent in places. The epiphyseal cartilages are normal, both in size and consistence. Microscopically, it is revealed that the process is confined entirely to the shaft of the bone, where the normal development and calcification of osteoid tissue is lacking. The formation of rows and their subsequent calcification and disintegration go on in a normal manner. It is at the stage of true bone-formation that the disease is manifest. The osteoblasts are diminished in numbers and deposit only a thin layer of osseous tissue. Calcification is thus delayed, deficient, or entirely absent. The other organs of the body are entirely normal.

Symptoms.—The general appearance of the newborn infant with osteogenesis imperfecta is characteristic. The skin and the subcutaneous tissue may be thickened ; on the other hand, they may be quite normal. The extremities are not shortened as the result of the cessation or retardation of growth, but are bent and deformed and may be the seat of multiple fractures. The ribs may be the seat of fracture. Some of these fractures may have united *in utero*, in which event we have the resulting deformity. Fractures may be so numerous as to give the long bones a nodular appearance. All the bones of the body partake of this fragility. The spinal column is soft and fragile, presenting anteroposterior and lateral deviations. The ribs may be fractured to an excessive degree. In Merkel's case no less than forty-three fractures were present. The clavicle shows fractures very similar to what is seen in cases of rachitis. The cranial bones show defective ossification, as is evidenced by the widely open sutures, or the cranial vault may consist simply of a membranous sac.

Dr. Nathan has published cases of osteogenesis imperfecta observed from infancy to advanced childhood or adolescence. He lays stress particularly upon the fact that the fractures in these children at first are scarcely observed. The slightest traumatism, such as a jar against some object, may produce these fractures. In some of his cases fractures occurred in the long bones, at various intervals, no less than thirty-five times. They occur soon after birth and may be present, though unsuspected, when the child is born.

When born, children are, as a rule, very weak and are carefully handled, for which reason fractures are not so likely to be observed at this time; as soon, however, as the general health improves and the children are allowed more liberty of motion fractures occur. They are attended with less pain and inflammation than in the normal individual, due, no doubt, to the slight traumatism. Union takes place rapidly and is usually firm. In some cases complete fracture does not occur, but infraction, resembling in a general way what is seen in rachitis. Some individuals not only survive childhood and learn to walk but may attain adolescence suffering from this disease.

Differential diagnosis must be made from chondrodystrophia fœtalis. In the latter disease the prognathous expression of the face is characteristic, with flattening of the nasal region; the bones, though shortened, are dense and hard, and, aside from slight bowing of the legs, are not deformed. In later life the chondrodystrophic individual is a dwarf, with shortened extremities and no predisposition of the bones to fracture.

Osteogenesis imperfecta is differentiated from rachitis by the absence of the rib rosary, the enlarged epiphyses, and other states characteristic of the disease. We can scarcely confound this disease with hereditary syphilis, or sarcoma or any new growth of the bone, or osteomyelitis. The dystrophy of syphilis is so characteristic as to bear no resemblance to the disease just described, the chief characteristics of which are fragility of the bones associated with defective ossification of the cranial bones.

The **etiology** of this disease is as yet a matter of speculation.

Its **treatment** must be founded on general indications, increasing the strength of the patient and protecting the bones from fracture.

IV. DIABETES MELLITUS.

Diabetes mellitus is of very rare occurrence in infancy and childhood. Simon says that he has met it in nurslings, but Monti doubts whether it can occur under the age of one year. In all his experience he has never seen such a case. Leroux, quoted by Monti, collected 147 cases of diabetes in children. The majority occurred between the fifth and tenth years. Of 159 cases collected by Saundby, 129 occurred between these years. Cotton has, in a recent article, shown that in children the ratio of deaths from diabetes to the whole death-rate is 0.04 per cent. in Chicago, and 1.2 per cent. in New York City.

The **etiology** of diabetes in children is practically the same as in the adult subject. Frerichs, Blanchard, Parry, and Roberts have shown that heredity plays an important rôle. In a case coming under my observation a sister of the patient had died of diabetes and four members of the family on the mother's side. In an instance reported by Roberts, 8 children of the family had died of it. It appears that in certain families there is a tendency to contract diabetes. There is no ground for assuming that diabetes in children follows traumatism or the infectious diseases, such as scarlet fever, measles, diphtheria, etc., any more frequently than in the adult. In some statistics, the sexes are shown to be equally affected. In others the disease is given as more prevalent in one or the other. Lemonnis has seen diabetes complicate congenital syphilis, tuberculosis of the lungs and of the mesenteric lymph-nodes. I have had a case complicated with tuberculosis of the mesenteric lymph-nodes.

The **symptoms** of diabetes in children, as given in the cases thus far published, do not extend over so great a period as in the adult. The cause of this must lie in the fact that there is a long period during which the symptoms are slight or escape notice. In a case which recently came under my care the child, nine years of age, showed symptoms only five months before she came under observation. At that time the mother noticed that the appetite was voracious and that there were great thirst and frequent urination. In spite of the large quantity of food and liquid taken, the child lost in weight. The amount of urine passed may be quite large. In Cotton's case it reached 104 ounces, in mine, 70 ounces daily. Monti has seen as much as 16 litres passed in twenty-four hours. Heubner and Hirschsprung found that the daily excretion of sugar may be from 30 to 113 grammes to the litre.

In most of the cases recorded there has been polydipsia. The skin is the seat of a lichen-like eruption which causes intolerable itching. Furuncles and boils are also of common occurrence. The urine may contain albumin, and hyaline and granular casts. In my case albumin was present, but no casts. There is as a rule constipation. The temperature may be normal or subnormal. If there is complicating tuberculosis, there will be a slight daily rise of temperature toward evening. In all the cases thus far published there was progressive emaciation. Acetone in the odor of the breath and diabetic coma preceded by intervals of delirium close the clinical course of the disease.

The methods of diagnosis do not vary from those pursued in the adult. The urine of a child suffering from polyuria, polydipsia, a voracious appetite, pruritus, and progressive emaciation, should be carefully examined for sugar. Infants who take foods such as malted milk, containing an enormous quantity of sugar, often show a temporary glycosuria, which should not be mistaken for true diabetes, and which is not attended by any of the clinical symptoms of that disease (Epstein, Koplik).

V. DIABETES INSIPIDUS.

(*Polyuria.*)

This is rare in infancy and childhood. If the daily amount of urine is three or more times the normal amount, there is polyuria. The specific gravity of the urine does not exceed 1006. Ebstein collected 10 cases in which the symptoms developed as a result of a cerebral inflammation in the vicinity of the fourth ventricle. The affection is sometimes hereditary. Cases have followed fright, the infectious diseases, meningitis, and traumatism. The cause is frequently obscure. The onset may be gradual or acute. Sometimes intense thirst or nervous symptoms usher in the disease. The nutrition may be maintained for years. The skin is dry, the body temperature below normal, and the symptoms do not differ from those manifested in the adult. The following case from my clinic was published by my assistant, Dr. Lewi:

Walter A., æt. seven years, was first seen at the dispensary. The family history was, for the most part, negative, except that three children had died of nervous diseases, one of them, aged three years, of spinal meningitis, and two others, when babies, of convulsions. The patient when a baby was healthy; he was breast-fed one year and had never had a convulsion. When two years old he had varicella, followed by pertussis; at the age of five he had measles, complicated with an obstinate conjunctivitis, but recovered. In October, 1892, while driving, he was thrown from a carriage in rapid motion, striking the right side of the head; no ill effects were noticed at the time. In January, 1893, he began to complain of pain in the back and in the nape of the neck. At about the same time it was noticed that he arose several times at night to urinate, and would invariably drink water after micturition; the mother noticed that he grew very nervous; the frequent micturition and increased thirst gradually became noticeable during the day, becoming so persistent that he was obliged to leave school. He was placed in a hospital, where he remained seven months; while there he lost flesh; none of the symptoms improved. He was on a rigorous milk diet during the entire time.

Status Præsens.—January 19, 1894, the child complains of pain on the right side of his head; says he feels chilly all the time and cannot stand still a moment. His face is pale and has an old person's look, with features sharp and pinched. The eyes are large and prominent, and the veins of the forehead dilated. The skin is exceedingly dry. The head is well shaped; careful palpation shows no sensitive spots. The chest is emaciated, with a slight rachitic girdle. The lungs, on auscultation give increase of voice-sounds at the right apex. The heart is normal, also the abdomen. The epiphyses of the ankles are enlarged. The glands at the angles of the jaw are enlarged, also those in the left axilla. *Urinary symptoms:* the child is passing a very large amount of urine; wakens on an average ten times a night to do so. The thirst varies with the amount of water passed; for the last few weeks he has complained of painful micturition. His appetite is excellent; he is on a milk diet. Weight is thirty-seven pounds; temperature (per mouth) 97.8° F. (36.5° C.). The urine examination was as follows: quantity in twenty-four hours, 6400 c.c., colorless; specific gravity 1.003; reaction acid, no albumin, no sugar. Microscopical examination negative.

A series of quantitative urea tests were made in this case. The general consensus of opinion is that in cases of diabetes insipidus the amount of solids, including the urea, is increased. The tests were made with the Doremus ureometer. A control test was always made. The table shows marked diminution in the amount of urea. In order to avoid error, fresh bromine was used.

Date.	Sp. grav.	Amount in 24 hours.	Urea.
Jan. 25.	1.003	6.300 c.c.	6.3 grammes.
Feb. 4.	1.003½	6.300 "	6.8 "
" 6.	1.005	5.200 "	7.2 "
" 8.	1.002	7.000 "	6.5 "
" 10.	1.004	5.500 "	6.8 "
" 17.	1.002½	7.500 "	7.8 "
" 24.	1.003	6.400 "	6.5 "
Mar. 18.	1.003	7.000 "	8. "
" 30.	1.003	7.300 "	7. "
Apr. 2.	1.003½	6.400 "	6.8 "

The treatment has been successful in some respects. The child was at once put on a general diet. Antipyrin was given. After the first few days there seemed to be an abatement of the nervous symptoms and slight diminution in polydipsia, but no permanent improvement. He was then given opium several weeks without result. Ergot was next given, and continued for about two months; under this treatment the pain on the right side disappeared; the restlessness became less, and the thirst likewise diminished. Under a generous diet the child has held his own; he still weighs thirty-seven pounds, his color is healthy, and the mucous membranes are normal.

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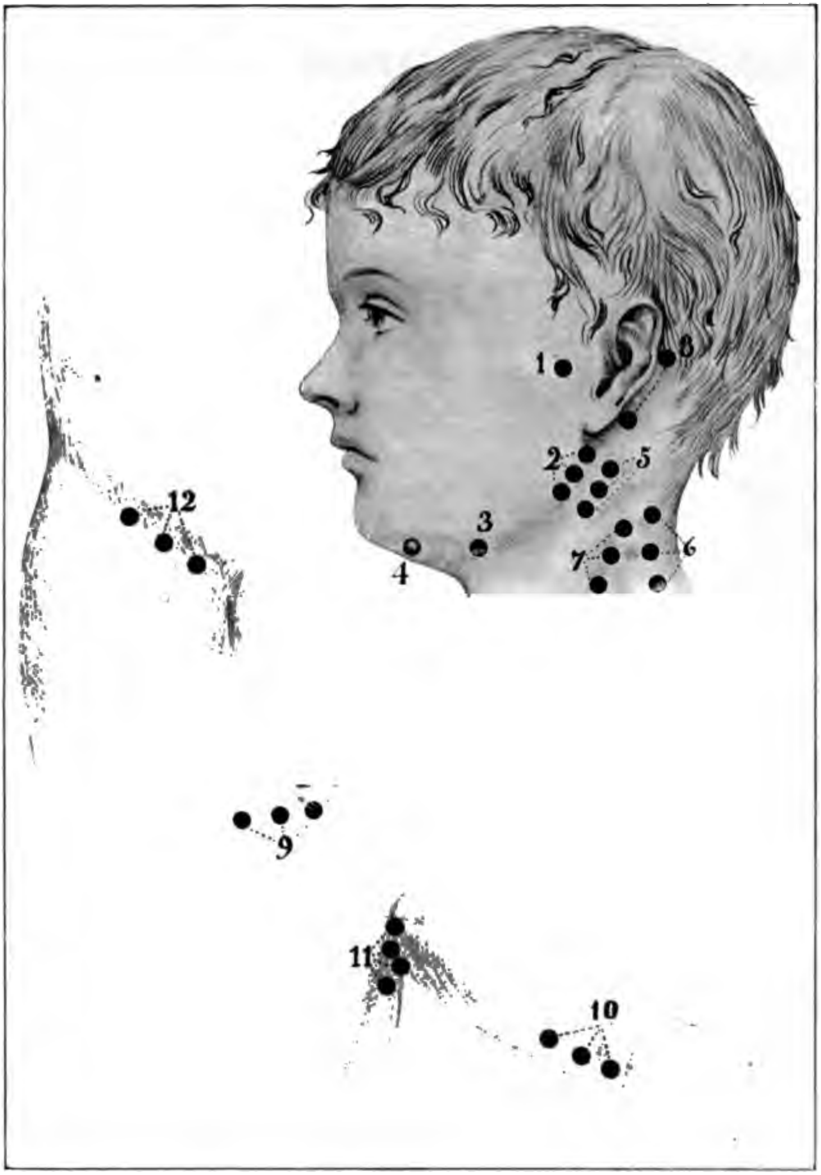
PLATE XXII.



Topography of Enlarged Lymph-nodes.

1. **Preauricular enlarged, with disease of the external auditory canal, or any eruption on the face, or parotitis.**
2. **Tonsillar nodes.**
3. **Submaxillary enlarged, with disease of the mouth, or skin eruptions over the lower jaw.**
4. **Submental enlarged, with chin eruptions.**
5. **Retropharyngeal enlarged, with infections of the pharynx and the retropharynx.**
6. **Nodes behind the border of the trapezius muscle enlarged, with disease of the scalp.**
7. **Nodes behind posterior border of the sternomastoid muscle enlarged, with infections of the retropharynx or the scalp.**
8. **Postauricular nodes enlarged, with mastoid disease or scalp infections.**
9. **Nodes above and behind the clavicle enlarged, with infections of the neck or mediastinum.**
10. **Nodes enlarged in infections of the hand or in eruptions such as those of syphilis.**
11. **Axillary enlarged, with infections of the arm, the axilla, and the upper chest.**
12. **Nodes of the inguinal region enlarged in infections of the lower extremity, syphilitic or other lesions of the genitals.**

SECTION IX.

DISEASES OF THE LYMPH-NODES, DUCTLESS GLANDS, AND THE BLOOD.

I. DISEASES OF THE LYMPH-NODES.

In any disease or irritation of the scalp the nodes of the neck may be enlarged behind the border of the sternomastoid. The onset of some diseases of infancy, such as rötheln or rubella, is indicated by slow enlargement of these glands. Infection of the tonsils will cause the lymph-nodes at the angle of the jaw to enlarge and sometimes to suppurate. In young infants and children, chronic enlargement of the tonsils with adenoids causes an enlargement of these nodes. Tuberculous glands may occur in this region. The post-auricular lymph-nodes enlarge in disease of the ear or of the adjácent parts of the scalp. Parotitis will cause a sympathetic swelling of the lymph-nodes in front of the parotid, and also below this gland at the angle of the jaw and beneath it.

Retropharyngeal adenitis will cause the nodes behind the pharynx to swell and to appear at either side of the neck in front of the border of the sternomastoid muscle.

Any eruption on the chin will cause an enlargement of the lymph-nodes from the tip of the chin to the hyoid bone.

Swelling at the angle of the jaw will frequently simulate parotitis.

In certain forms of congenital syphilis with mucous patches on the lips and at the angles of the mouth (rhagades) there is beneath the body of the jaw a symmetrical enlargement of the lymph-nodes of both sides (syphilitic adenopathies). The lymph-nodes of the groin will enlarge in balanitis of the prepuce, syphilis, tuberculosis of ritual circumcision, and also in eczema and intertrigo of the inguinal folds. The lymph-nodes of the femoral region will in infants and children enlarge or suppurate as a result of any infection of the foot, leg, or thigh.

In the later stages of tuberculosis, either of the lung or peritoneum, there may be a general enlargement of the nodes of the neck, axilla, groin, and elsewhere. In many infants and children of a lymphatic diathesis (lymphatism), the nodes of the neck and groin show slight enlargement. Such enlargements should not, in the absence of positive signs of tuberculosis elsewhere, be hastily pronounced tuberculous. After the exanthemata, the lymph-nodes of the neck, groin, and other regions may remain slightly enlarged. These enlargements usually retrograde to the normal in time, but if they remain rarely give rise to symptoms.

The physician should exclude every possible infection before concluding that an enlargement of the lymph-nodes in infancy and childhood is of a tuberculous nature. Cases of rachitis will show very slight enlargement of the lymph-nodes, especially in the inguinal regions. Forms of anæmia, such as von Jaksch's disease, also show these enlarged nodes. The lymph-nodes may be the seat of primary malignant disease, as in forms of lymphosarcomata. In malignant growths of the internal organs, such as the kidney, etc., they may be the seat of metastatic deposit. They are enlarged in acute and chronic forms of leukæmia and Hodgkin's disease. In these diseases the spleen and liver are also enlarged.

Acute Adenitis.

(*Acute Lymphadenitis.*)

The lymph-nodes in infants and children are peculiarly susceptible to acute infections, which are for the most part pyogenic (staphylococcic and streptococcic). Van Arsdale collected 500 cases of acute lymphadenitis seen in his experience. He found that 77 per cent. of them were in children. They are especially liable to the cervical infections. Eighty-five per cent. of the cases in children were infections of the lymph-nodes of the neck, the frequency in adults being only half as great.

Etiology.—Most of the infections of the lymph-nodes in children are, according to Van Arsdale, acute (79 per cent.). The majority of them are pyogenic. Children are subject to acute infections of the scalp, face, mouth, nose, tonsils, and mucous membrane of the nasopharynx. The lymph-nodes draining these regions are in the direct line of infection. Thus eczema and skin eruptions of all kinds, stomatitis of all varieties and inflammation of the tonsils and the nasopharyngeal space, will give rise to enlargement of the lymph-nodes. If the infection is severe, suppuration occurs. It is owing to these causes and to the breaches of surface caused by slight traumatism that this form of adenitis is so common. The essential exciting cause of acute lymphadenitis is the invasion of the nodes by pyogenic bacteria entering through the lymph-channels.

The **symptoms** of lymphadenitis in infants and children are essentially the same as in the adult subject. The node is at first felt as a hard nodular mass beneath the skin. One node or several may be infected. There is always some fever. At first the skin over the node is of normal color, but, as the inflammation progresses, it becomes involved, red, and finally, if not treated, there will develop all the signs of an ordinary abscess.

The **diagnosis** is not difficult. The history and general course at once point to the nature of the disease. When the region about the parotid is affected, it is at times difficult to tell whether there is an infectious parotitis, or whether the nodes just beneath or above the parotid are involved. A preauricular gland situated in front of the ear on the parotid gland is apt to enlarge and suppurate. The nodes underneath the angle of the jaw and in front of the border of the mastoid sometimes enlarge and suppurate, involving the parotid by collateral swelling. In all of these cases, it is important to remember that a line drawn parallel to the lower border of the body of the jaw marks off the parotid above, and the lymph-nodes below. In exceptional cases, the swelling of infectious parotitis may extend lower than this line.

The **treatment** of acute lymphadenitis is at first abortive. Cold applications to the nodes which are enlarged and accessible, such as those of the neck, relieve the pain and in many cases lessen the severity of the reaction. This result is frequently seen in cases where infection of the nodes of the neck results from tonsillitis. Sometimes, in spite of all that can be done, suppuration occurs as a result of infection of cervical, axillary (vaccination), and inguinal nodes. In that case, the affected node should be incised. The further treatment of such cases is surgical.

Chronic Lymphadenitis.

Chronic or subacute enlargement of the lymph-nodes in children may be pyogenic, tuberculous, or syphilitic. Of the cases collected by Van Arsdale, only 21 per cent. in infants and children were of chronic pyogenic origin, as against 12 per cent. in the adult. On the other hand, only 6 per cent. of all the cases of adenitis in infants and children were tuberculous. In the adult, the tuberculous forms of lymphadenitis are twice as frequent as in children. It is thus seen that even in chronic enlargements of the lymph-nodes of infants and children the occurrence of tuberculous forms gives the lowest percentage.

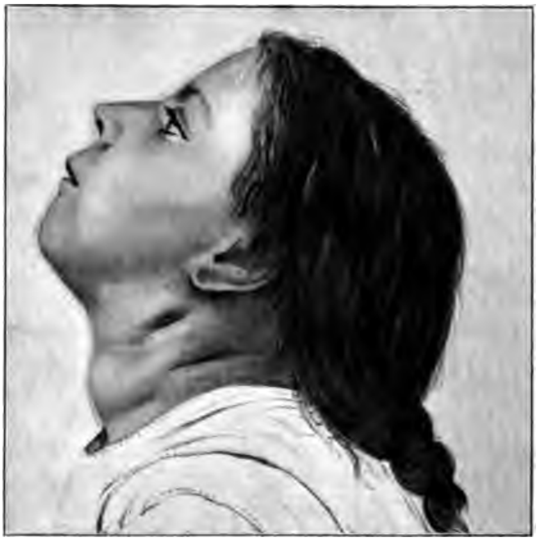
The **symptoms** of chronic enlargement of the lymph-nodes in infants and children are nodular tumors corresponding to the affected lymph-nodes. The enlargement may be single or multiple. Sometimes a whole packet of nodes is enlarged. The nodes most commonly enlarged are those at the angle of the jaw. This occurs in infants and children who suffer from chronically enlarged tonsils and adenoids. As a rule the nodes affected remain enlarged for months. At times they are somewhat less swollen. They do not suppurate unless there is a tendency to a breaking-down of tissue. In all of these cases there is not only toxic irritation, but also a true hyperplasia of the tissue of the glands. I have seen these nodes removed and opened. Some of them have a soft, broken-down centre resembling that of the tuberculous nodes.

The **treatment** of chronic lymphadenitis is directed toward removing the source of infection. If the tonsils are enlarged and adenoids are present, they should be removed. A tonic course of treatment, good food, out-of-door exercise, iron, and cod-liver oil is indicated. In spite of these measures many cases do not improve. If the enlargement of the nodes in such cases is localized, the question of the advisability of removing them arises. That measure should not be resorted to unless there is a reasonable certainty that they are tuberculous, and when all other treatment has failed.

II. DISEASES OF THE THYROID GLAND.

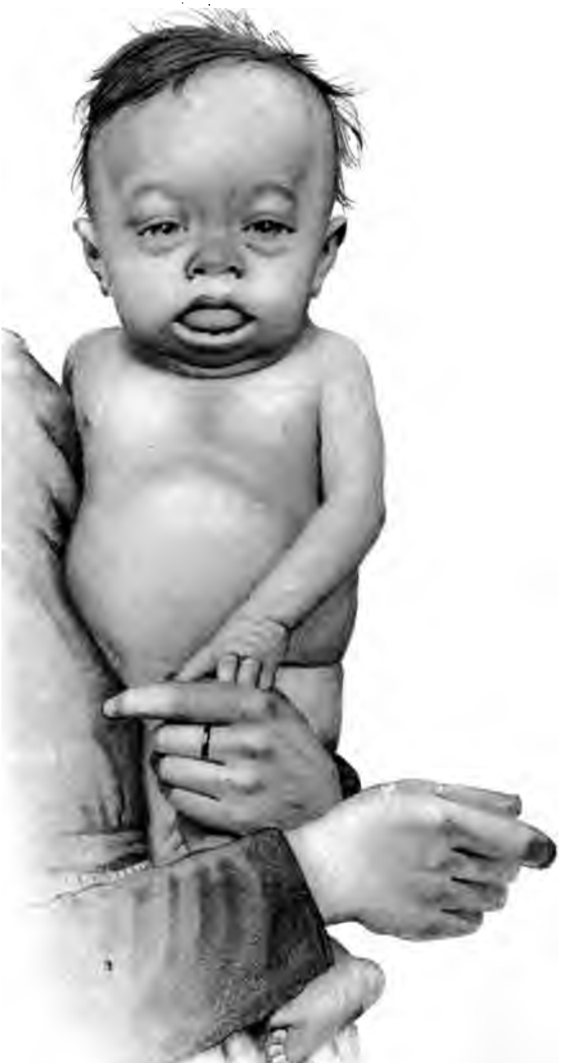
General enlargement of the thyroid is not uncommon in infancy and childhood. Normally the thyroid gland, and especially its isthmus, can be made out only by careful palpation. The isthmus is indicated by a very slightly raised structure passing across the trachea beneath the cricoid cartilage. The lateral lobes cannot be

FIG. 150



Enlarged thyroid in a child.

PLATE XXIII.



Sporadic Cretinism. Child fifteen months of age.

palpated. In cretinism and dwarfism, the enlarged lateral or supernumerary lobes beneath and just in front of the anterior border of the sternomastoid muscle can be palpated. Cystic growths of the thyroid are seen in front of the trachea, generally just above the notch of the sternum. They may occur in very young infants or in children of four or five years of age. Enlargement of the isthmus occurs chiefly in girls (Fig. 150). In these cases there is a disturbance of the heart functions and symptoms of the onset of morbus Basedowii.

Cretinism, Endemic and Sporadic.

Cretinism is a chronic affection which is characterized by a defective growth of the bones of the skeleton in their long axes, accompanied by a distinct set of mental symptoms and by changes in the soft parts.

Forms.—There are two forms, the endemic and the sporadic.

Endemic cretinism occurs in certain districts of Continental Europe. It does not exist in this country (Osler). The pictures presented by endemic and sporadic cretinism are similar. According to the recent studies of Dolega, His, and Bernard, their pathologic anatomy is also similar. Endemic cretinism is an advanced stage of a degeneration beginning with goitre manifestations. The resulting changes are due to "athyreosis," a suspension or disturbance of the functions of the thyroid gland. Sporadic cretinism, although also due to athyreosis, occurs without goitre. The peculiar formation of the skull in cretinism, endemic or sporadic, is now known not to be due to a premature synostosis of the os basilare and the sphenoid, as was at first thought by Virchow. The brachycephalic skull as manifested in a broadening of the bridge of the nose, and the prognathous expression is due to a deficient growth of the bones at the base of the skull, in their long axes. The sutures and fontanelles remain open for a long time. Dentition is delayed. The skin is myxœdematous in sporadic cretinism only. Dwarfism and anæmia are common to both forms.

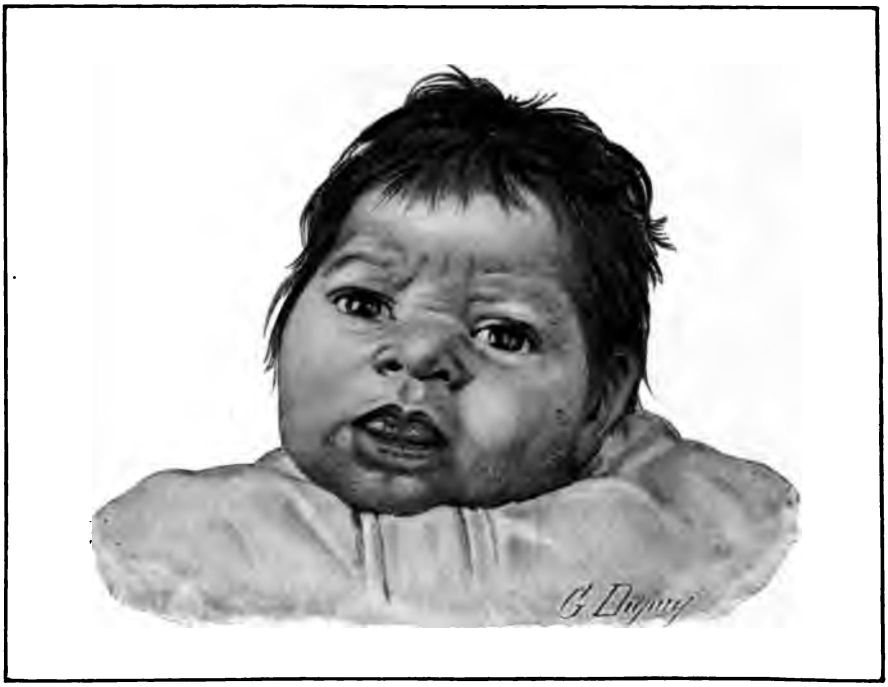
Sporadic Cretinism.

Occurrence.—The disease may appear in utero or at any time after birth. Fully one-half of the cases develop before the eighteenth month (Fletcher Beach).

Symptoms.—I have published cases in which the symptoms appeared within a month or five weeks after birth. The history was as follows: In one case there was another cretin in the family; in the others there was no such history. The birth as a rule was normal (Fig. 151). The infant was jaundiced, but fairly well nourished.

It lay in a torpid state and was only roused when severely teased. The infant was easily chilled. The cry was deep and coarse. The forehead was low and narrow. The eyelids were puffy. The tongue was large, broad, and thick, at times protruding from the mouth. The abdomen was large, and the thighs and legs were out of proportion to the length of the trunk. The skin had a greenish hue. The thyroid gland could not be found. The surface was cool and the rectal temperature 97° or 97.8° F. (36.1°–36.5° C.). The blood in these early cases has fœtal characteristics. There is no leucocytosis. In the cases which develop some months after birth the infant may at first be bright and normal. Six to nine months after birth, it may have had some slight illness, such as an adenitis, and after this the change was noticed, or the change may have occurred without any preceding illness. The infant ceases to notice objects about it, and becomes stupid and weaker. It may previously have attempted to walk or stand, but ceases to make an effort to do so (Plate XXIII.). The child's expression is idiotic. It has a meaningless smile most of the time and does not play. The skin has a wrinkled and myxœdematous appearance, the color being not

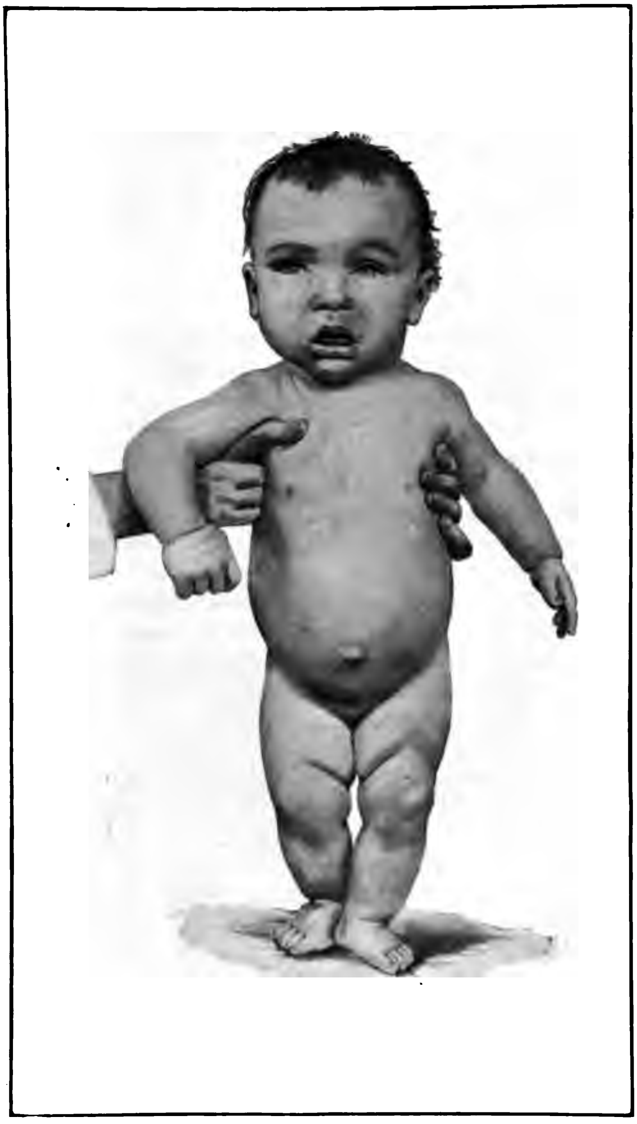
FIG. 151.



Congenital sporadic cretinism. Infant, four weeks old.

only pale, but also greenish. The nose is flattened, the lips are thickened, and the hair becomes dry and sparse. The forehead is narrow and the face has a prognathous expression—"monkey-like," as one mother expressed it. There are no teeth. The neck is short and thick. The genitals are large for the age. The skin

FIG. 152.

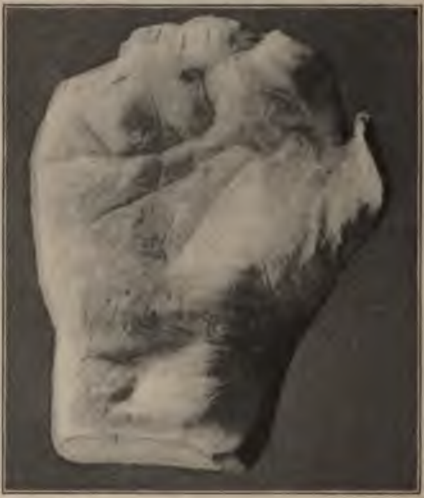


Sporadic cretinism; myxœdema marked. Child, twenty months of age.

of the scrotum is thickened. The anæmia in these cases is extreme. The hæmoglobin may be as low as 18 per cent. (Fleischl). The leucocytes may be as high as 18,000, and the red blood-cells 5,600,000.

In other cases, the symptoms are at first more nearly of the myxœdematous type. The skin, especially that of the face, has a greenish-yellow, waxy, puffy appearance. The upper and lower eyelids are swollen, as in nephritis. With these appearances, there are the dry hair, the macroglossia, the guttural voice, the dwarfish appearance, the protuberant abdomen, and the mental dulness. The expression of the face is less prognathous than in the first form. In one of my cases, the infant was in good health until the sixteenth month. It then developed abscesses over the body, after which cretinism set in (Fig. 152). The abscesses were peculiar, the granulations sluggish, and the pus was creamy. The skin was not œdematous but myxœdematous.

FIG. 153.

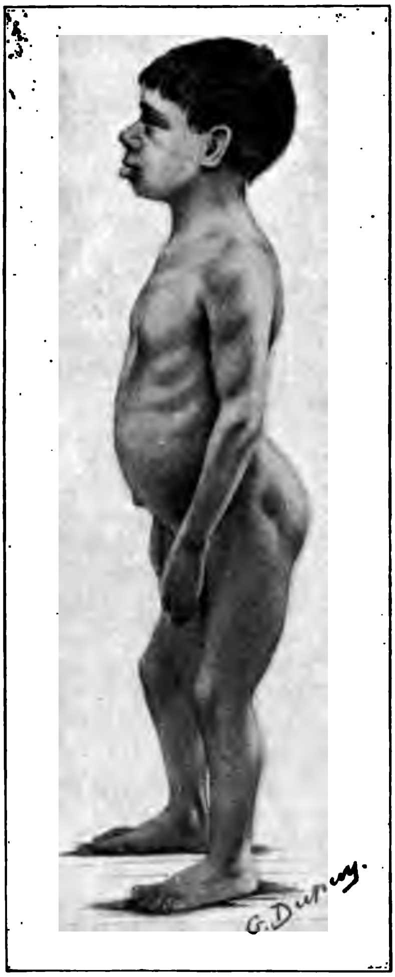


Cast of the hand of a boy cretin, four years of age. Flat and spade-like in form: It shows also the thickened and hypertrophied hypothenar eminence.

In both forms the hands are large, flat, and spade-like. The hypothenar eminence is thick, square, and hypertrophied, as in the lower animals (Koplik and Lichtenstein) (Fig. 153). In some cases the thyroid gland cannot be felt, in others it is small, and in exceptional cases there is goitre (7 cases of Osler's series). In some cases, supraclavicular masses of fat or fatty tumors behind the sternomastoid muscles are apparent. I have seen these masses of fat in the cases coming under my notice which had suffered a relapse after suspension of treatment.

The **etiology** of sporadic cretinism is as yet absolutely unknown. Experimental and operative pathology have demonstrated that interference with the function of the thyroid gland (athyreosis) will produce a condition (myxœdema) closely resembling cretinism (Horsely, Reverdin, Kocher). The essential cause of endemic cretinism is thought to be some form of infection (Fagge). Sporadic cretinism is also ranked by some authors among the infections.

FIG. 154.



Cretin, eighteen years of age (untreated).

Morbid Anatomy.—There are cases of sporadic cretinism in which the thyroid gland is absent. It has not developed in fœtal life and is not found at autopsy. In other cases there is found at autopsy a small atrophied gland which is sclerosed and much reduced in size. Such cases have been published as following the infectious diseases. Lastly, there are cases with goitre. The changes in the thyroid, when it is found in sporadic cretinism, have been described by Barker. There is an increase of connective tissue. The parenchyma is replaced by small and large irregularly shaped cells, which are granular and unlike the normal tissue. Some of the acini are almost solid; others are cystic and filled with colloid material. The cells may contain vacuoles; their nuclei may show "karyorrhexis." The nuclear changes are characteristic of degenerative processes. Some of the acini are replaced by connective tissue.

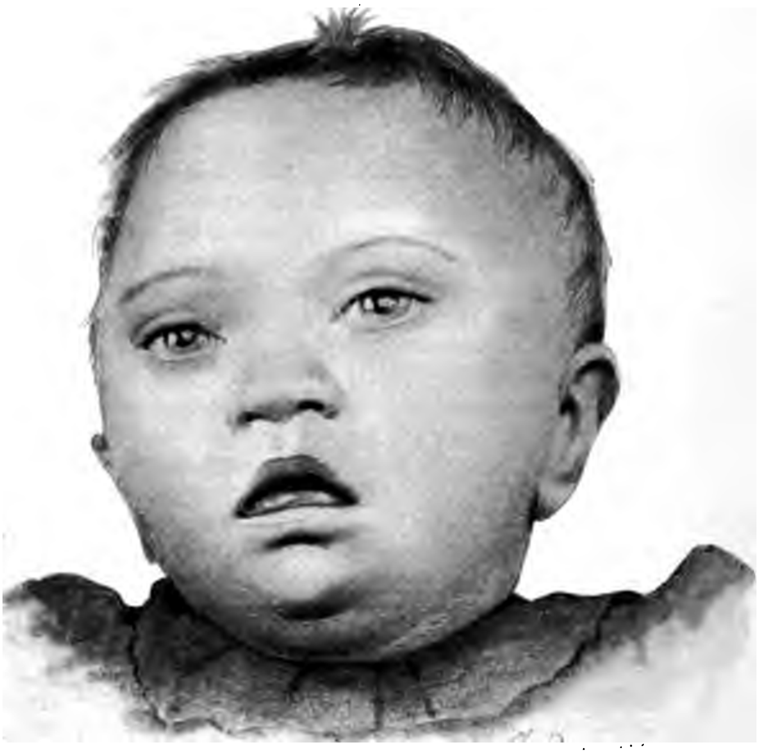
The Bones.—In the recent work of His, Dolega, and Bernard, it has been clearly shown that ossification in the pre-existent cartilaginous structures of the skeleton is delayed in all its phases. This is evinced in the delayed appearance of ossification centres, the delayed bony transformation of the epiphyses, and in the persistence of the epiphyseal zones. In some cretins, ossification is completed at a very late period of life; in others, infantile conditions are perpetuated. The dwarfing of the whole skeleton is thus explained, not by a premature synostosis, but by faulty proliferation and ossification of the epiphyseal cartilages. The bones of the skull are affected in the same manner as the vertebræ and the long bones, in that they fail to grow in their long diameters and in that ossification centres appear late.

The **diagnosis** is not difficult in advanced cases. The early cases require close study. In these, the stupidity increasing to absolute idiocy, the retarded growth, the change in the expression, the swollen eyelids, thick lips, dry hair, wrinkled myxœdematous skin, the flat, spade-like hands, the dwarfish appearance, and the reduced internal temperature, all point to the diagnosis. In later cases, the extreme anæmia, myxœdema, and pronounced prognathous expression of the face are apparent.

Sporadic cretinism must be differentiated from the following conditions:

Mongolian Idiocy.—This is a form of genetous idiocy with which cretinism is frequently confounded. The idiots resemble cretins. The growth is stunted. The mouth is kept open. The tongue is large and fissured; the papillæ of the tongue are enlarged and erect. The tongue protrudes from the mouth (Plate XXIV.); the lips are thick; the voice is coarse and guttural. The temperature may be subnormal, but is generally normal. The skin is dry and the hair coarse. In young infants the skin may be delicate. The patients are easily chilled. The musculature is flabby. The infants cannot hold the head erect. The occiput is flattened, the neck short and thick.

PLATE XXIV.



Mongolian Type of Idiocy. Child two years of age.

There is strabismus, and the axes of the eyelids have a Mongolian slant—that is to say, they converge. The inner eyelid comes down toward the nose with a rapid slope. The bridge of the nose is flat. The head is small and obtusely rounded; the antero-posterior diameter is nearly equal to the lateral one. The fontanelles remain open late. The skin, however, is not myxœdematous, nor is the expression prognathous as in the cretin. The anæmia is as a rule marked; in some cases the skin has a greenish hue. There is a curving inward of the tip of the little finger. The second phalanx is short and the terminal phalanx displaced. West has shown that although this deformity is very common in these idiots, it is not pathognomonic of Mongolian idiocy. Many of the subjects grow to adult life and have some degree of intelligence.

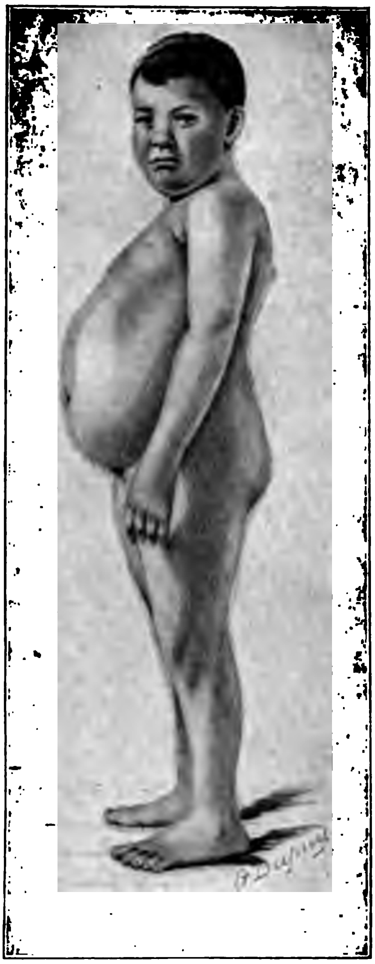
The Dwarf with Idiocy.—There may be several of these dwarfs in a family. The thyroid gland is enlarged at the beginning or during the course of the condition. The mental state is much stunted. The general growth of the body is retarded. The dwarfs are, however, well formed. The hands and extremities are perfect and the skin is not myxœdematous.

Infantilism combined with lipomatosis may be confounded with cretinism. In this form of disease there is no myxœdema and the skin is very delicate and soft. The genitals are atrophied. The expression of the face is that of child-like simplicity, the forehead is low and narrow. The hair is dry, and does not grow; the finger-nails do no grow. There may be, as in the case I published, blindness. The mental state is one of mild idiocy (Fig. 155).

The **treatment** of cretinism constitutes one of the most marvellous chapters of modern medicine. The administration of thyroid extract results in a partial restoration of the mental capacity and a return to growth and development approaching the normal. I published in 1897 some cases of cretinism diagnosed early in infancy, in which the treatment was begun at once. In those in which the treatment was begun at the age of one month, the children have become bright and apparently normal. In those in which it was inaugurated at the fifteenth month, the children have, after five years of treatment, remained somewhat backward in mental development. One patient, now a boy of six years, goes to school and recites his alphabet, but is very simple in manner. In these late cases the treatment does not give the complete results at first expected.

I begin treatment by the administration of the dried extract, grain ½ (0.03) t. i. d., and increase the dose until the infant takes grain j (0.06) three times daily. After the symptoms have retrograded, the dosage is kept stationary for a few months. It is then reduced or the remedy is given only every other day. If symptoms, such as stupidity, pallor, or reduced temperature reappear, the dose is increased. The first sign of improvement is a reduction of the anæmia, as evidenced in the increase of hæmoglobin. The body temperature rises to the normal. The skin becomes of normal delicacy and supple. The stature increases and the hair becomes glossy. Thomson, of Edinburgh, has published cases of adult cretins whose bones became softened after the prolonged administration of thyroids. These were cases in which treatment was begun late in life. The symptoms of excessive administration of thyroids include rise of temperature and slight diarrhœa.

FIG. 155.



Infantilism and lipomatosis universalis in a boy ten years of age.

I have found thyroid therapy of doubtful utility in cases of Mongolian idiocy. In the dwarfs above mentioned, it causes increase of stature; the intelligence, however, remains backward.

III. DISEASES OF THE THYMUS GLAND.

Landmarks.—The thymus is a glandular organ enclosed in a capsule. It is situated in the anterior mediastinum, and contains in its structures a white tenacious fluid substance which is present in varying quantities. Sappey shows that the thymus in the newborn infant extends from the upper edge of the manubrium sterni, 5 cm. downward. Its upper border may reach the isthmus of the thyroid or may be removed 2½ cm. from it. It extends downward to the middle or upper third of the pericardium. In exceptional cases it may have a longitudinal diameter of 11½ cm., reaching the diaphragm (Triesethau). The thymus is about 2 to 3 cm. wide. Luschka makes it unsymmetrical, consisting of two lobes united by an isthmus. It lies over the course of the pulmonary artery and is surrounded by a reflection of the pericardium. It is separated from the sternum by loose connective tissue. Its length varies from 4 cm. in the nursling, to 11 cm. in the ninth year, the average ratio to the body length being 1 to 7 or 8.

Weight.—Its weight varies. In the results which I obtained in collaboration with Jacobi, it did so within wide limits. In infancy the average weight is 20 grammes; from the second to the fourteenth year it is 24 grammes. After the twenty-fifth year the thymus atrophies and may weigh 2.2 grammes (Friedeleben). In abnormal states the weight may be 32 grammes (Triesethau, Pott). The causes of the enlargement of the gland and the conditions under which it occurs are not as yet known. The gland is large in infants dying of the most diverse diseases.

Percussion.—Under the most favorable conditions it is difficult to ascertain the exact size. The thymus has sometimes been marked out as large during life, and post mortem found to be small. As a rule, an area of dulness situated behind the upper part of the sternum, and discernible on gentle percussion, may be cautiously interpreted as due to the thymus (Sahli). An unsymmetrical area giving dulness on one side of the sternum is probably due to the thymus (Luschka), especially in subjects under the second year. The thymus may be seen by *x*-ray as a shadow behind the upper sternal region.

Abnormal Conditions.—None of the abnormal conditions of the thymus can be diagnosed with certainty during life.

Hypertrophy of the Thymus Gland, Including So-called "Thymus Death."

Simple hypertrophy of the thymus gland, irrespective of its presence as a cause of sudden death, has been observed by Virchow, Grawitz, Jacobi, and others. It may exist without causing any symptoms, and only be discovered post mortem in children who have died of various diseases. In other cases an enlarged or hypertrophied thymus has been described as causing a series of symptoms similar to what is seen in the adult subject in forms of asthma. Virchow, Grawitz, West, and Goodhardt have described such cases under the head of "Thymic Asthma." These cases are attended with paroxysms resembling those of laryngismus stridulus with difficult breathing. Some of the cases described by the above authorities have eventuated in convulsions and sudden death. Recently Hochsinger has attempted to revive the term "thymic asthma" as applying to cases of laryngeal stridor; the symptom-complex in such cases being due, in his opinion, to an enlarged condition of the thymus.

There has been much discussion as to the existence of such an entity as "thymic asthma."

There is another form of sudden death, the so-called "thymus death," which has been ascribed to hypertrophy of the thymus gland. These cases have been described by Virchow, Grawitz, Pott, and others, and there seems to be a tendency in some quarters to attribute certain cases of sudden death to the existence of an enlarged thymus. In one case, described by Pott, the thymus weighed 32 grammes, was 9 cm. long and 1½ cm. thick. Cases of thymus death have been described, for the most part, in children who are the victims of a condition known as status lymphaticus. This condition should be differentiated from that described under the heading of Scrofulosis, and for the sake of clearness will be described under the head of Status Lymphaticus combined with that of thymus death.

In the work of Jacobi it was shown that hemorrhages of the thymus are not uncommon, and are present in a number of conditions, especially in pertussis. Inflammation of the thymus may be present in inflammatory conditions of the pleura and pericardium. Steudener has published a case of sarcoma of the thymus, and Vogel one of carcinoma of that organ, occurring in childhood. Demme published a case of isolated tuberculosis of the thymus. In the monograph of Jacobi, general tuberculous infection of the thymus was investigated, as was also the condition as found in diphtheria. In the latter disease necrobiosis of the thymus was found as described by Oertel in other organs. Congenital syphilis may manifest itself in arterial and connective-tissue changes. Abscess of the thymus is rare.

Status Lymphaticus.

(*Lymphatism; Lymphatic Constitution.*)

This condition is found chiefly in children who are subjects of rachitis and are moderately well nourished but anæmic. They have enlarged lymph-nodes at the angle of the jaw, in the axilla, and in the groin, and may have attacks of laryngismus stridulus. They have enlarged tonsils, adenoid tissue in the posterior nares, and enlargement of the adenoid tissue at the base of the tongue. On the other hand, they present none of the skin-, bone-, and joint-affections seen in the scrofulous or tuberculous subject. Escherich has published cases in which there were 30 attacks of laryngospasm a day. The patients also have symptoms of increased excitability of the peripheral motor nerves, such as Trousseau's phenomena and Chvostek's symptom. I have had one case in which there was an attack of laryngismus at every crying-spell. The patients are in constant danger of sudden death. In rare cases in which sudden death has occurred an enlarged thymus has been found, and other lesions which will now be described under the title of Thymus Death.

Thymus Death.

There are two distinct sets of cases of sudden death in which the thymus has been found to be enlarged. The first are those in which, post mortem, absolutely no other change has been found than the presence of an enlarged thymus. In these cases the viscera were said to be absolutely normal, but, as has been stated elsewhere, there were evidences of lymphatism, such as enlarged tonsils, lymph-nodes, and solitary follicles in the intestine.

The second set of cases is that in which the thymus was found not only to be enlarged, but apparently pressing on the trachea or arch of the aorta, causing complete obliteration of these organs. The latter set of cases were recorded by Beneke, Lange, and Weigert. But few of these cases published are to be considered in the category of thymus death, for these rather represent pathological growths of the thymus similar to any other tumor which might lead to pressure effects. Such a condition of the thymus is exceedingly rare. What interests the physician most, especially as the cause of sudden death, are the cases of enlarged thymus in which, as in the first set, no signs of pressure were found, either on the large vessels or the bronchi. That death in these cases is not caused by pressure is now generally conceded. The theory advanced by Paltauf and Escherich is not unreservedly accepted by all. Paltauf contends that the sudden death is due to an anomalous lymphato-chlorotic constitution, the enlarged thymus thus being only one of the manifestations of a general disturbance of nutrition, in which we also find enlarged lymph-nodes and tonsils, and hyperplasia of lymphatic tissue. Under the influence of this condition there are changes in the nerve-centres of the heart, as a consequence of which the least excitement may result in fatal paralysis. Escherich, in addition, while accepting this theory, thinks that in the condition of lymphatism there is an auto-intoxication whereby the nervous system is in a state of morbid irritability and instability which results in heart-syncope. In this condition the functions of the thymus are probably disturbed, much like that of the thyroid in myxœdema or Basedow's disease.

On the other hand, Richter, in a recent paper, which has been referred to in the section on Sudden Death, has analyzed all the cases published of the so-called thymus death. In most of these cases, in his opinion, there were present anatomically other conditions, such as bronchitis, intestinal cataarh, or some other disease, to account for the fatal issue. In most children overtaken by this form of death there is a condition of lymphatism, and this, in addition to the growing thymus, which at the age of two years is quite large, has been made accountable for the death of these infants and children, whereas close study will always reveal some other morbid condition fully equal to causing this issue.

Thymus death is one of the rarer forms of sudden death in early infancy, as compared with other forms. I have seen it twice, and know of nothing more distressing than such an occurrence. The physician may be examining such a child for a slight ailment, when suddenly the infant throws the head backward, there is a noiseless or snappy inspiration, the eyes turn upward and sideways, the pupils dilate, there is cyanosis both of the face and tongue as the latter becomes swollen and caught in the jaw; there is a convulsive contraction of the body backward. There are several inefficient, noiseless, shallow inspiratory movements, the body then relaxes, the face becomes ashy pale, and the infant, within one or two minutes, is dead. The heart ceases to beat at the beginning of the attack. It is really a syncopal death.

There is another form of death in lymphatic infants and children which occurs in chloroform narcosis. In such cases the heart may suddenly cease to beat during the narcosis; or, as in one of my cases, the child may have withstood the narcosis, though it was noticed to have taken the chloroform badly. Twelve hours after the operation —which in this case was one of appendicitis—the temperature rose slightly, there was a rapid increase in the heart action, the pulse mounting in a short time so that it could no longer be counted; while the heart beat at the rate of over 200 a minute (cardiac paralysis); the pulse could not be felt at the wrist. Death occurred with all the signs of paralysis of the cardiac ganglia.

In the case last described the child was extremely lymphatic, had a thymus enlarged to percussion, and a year previous had been operated on for adenoid vegetations and enlarged tonsils. The lymphatic nodes throughout the whole body were enlarged. The appendicitis from which the child suffered was one of the mild catarrhal type. There was no septic peritonitis.

Treatment.—Inasmuch as death supervenes in these cases before anything can be done in an orderly way, it is almost superfluous to speak of treatment. Pott, however, and others have performed tracheotomy in these cases with a view not only of relieving the spasm of the glottis, which in some instances is present, but of performing artificial respiration. Others have intubated. In those cases in which the heart has ceased to beat, we can scarcely expect to revive its action. In one case of a lymphatic child in which the heart failed at the outset of the chloroform narcosis, became irregular, and threatened to stop beating, artificial respiration, the Laborde method of resuscitation, and massage over the cardiac area according to the method described by Maas, brought the child to life again. We will not always succeed in this manner.

The treatment of the general condition—the status lymphaticus—consists in the removal of the enlarged tonsils and adenoids. In these cases the condition of the lymphatic-node enlargements is vastly improved by the operation. Good food, cod-liver oil, and the preparations of the iodide of iron are also indicated.

IV. DISEASES OF THE SPLEEN.

Anatomical.—At different periods of childhood the length of the spleen varies from 4 to 10 cm., the breadth from 2 to 5 cm., the average thickness being about 0.5 cm. It forms an oval-shaped body, behind the ninth, tenth, and eleventh ribs, the long axis running in the direction of the ribs. Up to the second month of life, the anterior edge of the spleen is found in the midaxillary line; after that, it may be found further forward than this line, or posteriorly to it. The upper edge corresponds to the upper edge of the ninth rib; the lower border to the lower border of the eleventh rib. The spleen may be located by percussion and palpation.

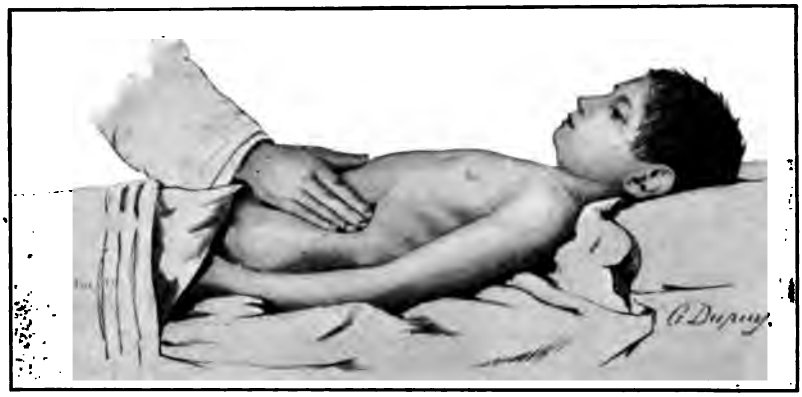
Percussion.—The patient is caused to lie on the back. It is not necessary to cause children to lie in an inclined lateral posture. The upper border is first located by percussing from above downward in the midaxillary line on the left side. At the seventh rib is a strip of slight dulness extending from the seventh to the ninth rib. I have been able to locate it in infants and in children under the age of six years. There can be no question as to its existence, although there may be doubt as to its causation. Symmington, in his frozen section, shows that, in a girl six years of age, the left lobe of the liver is distinctly on the left side behind the seventh and ninth ribs. Sahli ascribes the strip to what he calls the deep dulness of the spleen. From the ninth rib downward, there is absolute dulness, then flatness, due to the presence of the spleen proper behind the chest wall. The anterior border of the spleen is located by percussing in a horizontal direction toward the axillary line along the tenth rib.

Palpation.—The enlarged spleen can be distinctly made out by palpation. The abdomen should be relaxed. It is sometimes necessary to flex the thighs slightly, in order to relax the abdomen. In young infants it is not necessary.

The physician stands at the *right side* of the patient and with the palmar surface of the fingers of the right hand palpates the abdominal parietes just beneath the border of the ribs (Fig. 156). As the patient inspires deeply, the hand is by steady pressure insinuated beneath the ribs in an upward and backward direction. In the vast majority of cases under the tenth year, the normal spleen may thus be felt.

In practice, it may safely be said that a spleen which cannot be felt below the border of the ribs is not enlarged, unless some condition, such as the presence of fluid or tympanites, prevents thorough palpation. I have rarely failed to palpate the enlarged spleen satisfactorily. Enlargement of the spleen is found in rachitis, chronic gastro-enteritis, sepsis, typhoid fever, malarial fever, varicella, syphilis, anæmia infantum pseudoleukæmica, leukæmia, Hodgkin's disease, congenital syphilis, cirrhosis of the liver, amyloid degeneration, heart disease, and simple catarrhal jaundice.

FIG. 156.



Method of palpating the spleen.

From these statements it will be seen that enlargement of the spleen in infancy and childhood is pathognomonic of no one disease, and should not lead to any one conclusion. It is only corroborative in the presence of other signs and symptoms. Without a very thorough and painstaking examination of the blood, the significance of the enlarged spleen in the febrile and afebrile affections cannot be determined. In enlargements of the spleen such as are met in rachitis, heart disease, syphilis, chronic gastro-enteritis, icterus, varicella, examination of the blood may not be necessary.

Splenic and Kidney Tumors.

In rare cases in which sarcoma of the left kidney is suspected, it may be necessary to exclude tumor of the spleen.

An enlarged spleen is smooth on the surface and has a sharp anterior edge interrupted by an indentation—the hilus. The tumor is pointed and sharp below. It can be grasped deep in the lumbar region behind.

Kidney tumors are irregular on the surface and marked out into lobes, some of which may be cystic. The tumor projects upward behind into the lower part of the chest. The whole lumbar region is flat on percussion. The borders of the tumor are rounded. On the other hand, I have made an autopsy in a case of cirrhosis of the liver and spleen in which the latter organ during life showed uneven tumors on its surface (gummata). The physician must be partly guided by the history of a case. The urine should be examined in cases of sarcoma of the kidney, and the blood in cases of enlarged spleen. I have seen a subphrenic abscess displace the spleen downward. The left lobe of the liver was also displaced in the same direction. Under anæsthesia, a round mass could be felt above the spleen, which was enlarged. Behind, the lung came well down to the bottom of the chest, as was evinced by the presence of the respiratory murmur. Dulness was, however, present in the left axillary line and behind. On exploratory puncture in the posterior axillary line, the subphrenic abscess was found to be present.

V. DISEASES OF THE BLOOD.

Leading General Characteristics of the Blood in Infancy and Childhood.

For diagnostic purposes, it is important to bear in mind certain characteristics of the blood in infancy and childhood. Ehrlich has shown that conditions normal to the blood in early life are of grave import if found in the adult.

The Red Blood-cells—the Erythrocytes.—During the first three days of life, nucleated red blood-cells are found in the normal blood. In the newly born infant, the red blood-cells number from 4,500,000 to 6,500,000 to the cubic millimetre (Hayem). There is a polycythæmia. This condition is found during the first few days of life. On the fourteenth day there is an average of 5,500,000 red blood-cells to the cubic millimetre. From the second to the tenth year the average number is 5,000,000 (Otto, Schiff, Sörenson). The polycythæmia in the newly born infant is greater if the tying of the umbilical cord is delayed until its pulsations cease. Weaklings show a diminished number of red blood-cells. In addition to imperfect nutrition, anæmia of any kind, acute or chronic cachexia, and certain drugs, such as antipyrin, antifebrin, phenacetin, and lactophenin, reduce the number of red blood-cells by disintegrating a certain proportion of them (Monti). Infectious diseases, such as malaria, scarlet fever, typhoid fever, and sepsis, have a similar influence. In severe anæmia, such as that accompanying rachitis, nucleated red blood-cells appear in the blood. These are also found in the severe primary anæmias, in acute leukæmia, and in pernicious anæmia of infants and children.

The White Blood-cells—the Leucocytes.—The number of leucocytes in the newly born infant is high, being from 18,000 to 30,000 to the cubic millimetre (Hayem, Guppen). It gradually falls to 12,000 to the cubic millimetre, the average for infants. The percentage of lymphocytes is at first small in comparison with that of the polynuclear leucocytes. Gundobin, whose work has been confirmed by Carstanjen, found that the polynuclear leucocytes preponderate in the newborn infant. They increase and reach their highest figure in the first forty-eight hours of life. They then diminish in number, while the mononuclear lymphocytes increase proportionately until the seventh or tenth day, when the blood assumes the characteristics which distinguish it during the period of infancy. During infancy the mononuclear lymphocytes are more numerous than the polymorphonuclear leucocytes. The following table is taken from Gundobin's figures:

	Polymorphonuclear leucocytes.	Mononuclear lymphocytes.	Transitional forms.
Immediately after birth . .	63 per cent.	25 per cent.	12 per cent.
Forty-eight hours after birth.	70 "	21 "	19 "
Infancy	34.6 "	59 "	6.4 "

In normal infants and young children, the number of leucocytes to the cubic millimetre may vary from 13,000 to 20,000 (Japha). The so-called digestive leucocytosis found in the adult is inconstant in infants and young children (Japha). There is undoubtedly an inflammatory leucocytosis in infants and children similar to that seen in the adult. It occurs in pneumonia, scarlet fever, rheumatism, sepsis, diphtheria, post-hemorrhagic anæmia, and cachexia (sarcoma). In the normal state, the leucocytes may reach a minimum of 6000 to the cubic millimetre (Monti). This fact should be borne in mind in estimating the leucopœnia in typhoid fever, malaria, tuberculosis, and in other infectious or toxic states.

The transitional forms of leucocytes are numerous in the newly born infant, reaching their maximum from the sixth to the ninth day. The eosinophiles are present in the same number as in later life (Japha).

The Hæmoglobin.—The blood is richer in hæmoglobin at birth than later in life (Morse, Leichtenstern, Rotch). After birth the percentage of hæmoglobin sinks, and at the third month reaches that of later life. Carstanjen found the hæmoglobin on the average 100 per cent. up to the twelfth day. The lowest percentages are found from the sixth month to the second year. There is, in exceptional cases in normal children, a very high percentage from the fifth to the tenth year, ranging from 95 to 110 (Widowitz, Leichtenstern, Hock, and Schlessinger). The percentage in healthy children may be as low as 60 (Fleischl) or 8.4 grammes to 100 c.c. of blood. At the third month of infancy it may range from 69 to 94; up to the second year it may range from 62 to 81 (Monti). There seems to be no fixed normal limit. Anæmia or toxæmia of any kind and infectious diseases diminish the hæmoglobin.

The Specific Gravity.—The exact clinical significance of the specific gravity of the blood is little understood. The specific gravity is high in the newly born infant, ranging from 1.056 to 1.066. From the sixth month to the tenth year it varies from 1.050 to 1.056 (Monti). These figures correspond to those of Hock, Schlessinger, Lloyd, Jones, and others. The blood of strong children and breast-fed infants has a higher specific gravity. Diarrhœa may raise it, but rarely to a ratio of more than 0.004 part per 1000. The specific gravity is increased in the infectious diseases, pneumonia, pleuritis, endocarditis, typhoid fever, and tuberculous meningitis, and falls on the decline of these processes. It is also increased in congenital heart disease, chorea with endocarditis, icterus, and diphtheria. It diminishes with the loss in weight accompanying anæmia and nephritis, and in cachexia (Hock, Schlessinger, Monti, Hammersley, and Felsenthal).

Anæmia.

Anæmia is a condition resulting from a deficiency in the blood of one or more of its constituent elements. It may be either congenital or acquired. In the latter case it may either be secondary to other conditions or occur as a primary disease. Congenital anæmia is seen at birth in infants born of badly nourished mothers, who during pregnancy have suffered from some disease of the placenta, or from syphilis, tuberculosis, or malaria. The fœtus in utero becomes anæmic. Acquired anæmia appears after birth. It is either secondary to some acute loss of blood (post-hemorrhagic), to chronic loss of blood, or is caused by defective nutrition, unhygienic surroundings, diseases of the various organs, toxæmia, infectious diseases, or parasites.

Primary or essential anæmia is the form in which the changes in the blood play so important a rôle that it is assumed there is a disease of the blood itself or of the blood-forming organs (Monti). Such are the forms of leukæmia, chlorosis, and pernicious anæmia.

Simple Anæmia.

(*Secondary Anæmia.*)

Secondary simple anæmia may follow some acute or chronic loss of blood. In acute post-hemorrhagic anæmia, the increase of fluid elements keeps pace with the loss of blood if the loss, though small, is repeated at short intervals. Children show the effects of loss of blood much more quickly than adults. Hydræmia is the condition which results when the loss is marked. The fluid elements increase, and there is a diminution in the specific gravity of the blood and in the amount of hæmoglobin. Hydræmia may result in children without hemorrhage; that is to say, it may occur in extreme severe anæmia secondary to some disturbance of nutrition or to illness. In post-hemorrhagic anæmia the coagulability of the blood is increased immediately after the hemorrhage. Ehrlich supposes this to be due to an increase in the number of blood-plates. After the hemorrhage, the regeneration of blood is in the infant, as in the adult, indicated by the formation or appearance in the blood of microcytes, megalocytes, and nucleated red blood-cells (normoblasts). The severe forms of this variety of anæmia also show polychromatophilic properties of the red blood-cells. These are so poor in hæmoglobin that with various stains the normal reaction is very much changed. There are various shades of the stained red blood-cells. In recent and severe cases of post-hemorrhagic anæmia there may be leucocytosis. There is an increase of the polynuclear neutrophilic leucocytes (Monti, Ehrlich). Nucleated red blood-cells (normoblasts) may appear in severe cases. Poikilocytosis is also one of the changes seen in the blood.

Secondary anæmia of a mild or of a severe type is also seen in infants and children who suffer from defective nutrition. It complicates or accompanies rachitis, syphilis, scrofula, tuberculosis, gastro-intestinal catarrh, chronic endocarditis, purpura, morbus Werlhofii, and infectious diseases.

The **symptoms** of mild anæmia in infants and children do not differ materially from those of adults. The patient is pale and the mucous membranes have a characteristic pallor. The appetite is capricious. The patients also suffer from symptoms due to the primary affection—syphilis, rachitis, acute infectious disease, gastro-enteric disturbance (acute or chronic), or cardiac affection. The pallor of cardiac disease or nephritis is characteristic in infants and children, as in the adult.

The anæmia if of a severe type takes the hydræmic form. In the severer forms of anæmia, especially in infants and very young children who suffer from syphilis or rachitis, the skin is waxy white or yellowish. The ears are absolutely devoid of any color of blood. In cretinism the skin has a greenish-yellow hue. Infants do not show the symptoms, such as dyspnœa or palpitation, seen in older children on exertion. The muscles are flabby and there is a disposition to lie quietly in the crib. The spleen may be large, and the liver also, especially if rachitis or syphilis is present. In cases in which the anæmia is extreme, the spleen may be normal.

Infants and very young children do not always show the anæmic murmurs which are heard over the heart area in older children. In older children murmurs of that variety may be present with a venous hum in the neck, and the symptoms of mild and severe anæmia are essentially those of later life. These are indisposition to exertion, feelings of weakness, drowsiness, lack of appetite, irritability, and restlessness. Some of the severe forms of anæmia show for weeks a very slight irregular febrile curve. In many cases the fever is due to intestinal toxæmia.

The Blood.—The mild forms of simple anæmia may show only a diminution in the amount of hæmoglobin, a very slight diminution in the number of red cells, a reduction of the specific gravity, and if there is a primary affection which, like pneumonia, causes an increase in the number of leucocytes, leucocytosis. My records of severe forms of anæmia in infants and young children show a diminution in the amount of hæmoglobin (18 per cent.). The blood shows microcytes, megalocytes, megaloblasts, and normoblasts. Increase of mononuclear lymphocytes is proportionate to that of the polynuclear leucocytes. Poikilocytosis in various forms is present, as are also polychromatophilic phenomena. In the severe forms of anæmia due to malarial poisoning I found, in addition to the plasmodium, microcytes, megalocytes, and megaloblasts. The eosinophiles are not increased. In severe anæmia, the physical characteristics of the blood are striking. It may be so thin as to separate on puncture into a reddish and a colorless portion resembling beef-water.

Chlorosis.

Chlorosis is a form of primary anæmia. It is not a disease of infancy or childhood, and is mentioned here only in order to complete the classification of diseases of the blood. Its etiology is obscure. Virchow believed it to be due to congenital narrowness of the whole arterial system and smallness of the heart. This theory does not explain the cases in which recovery takes place. Meinert ascribed the condition to an irritation of the abdominal sympathetic. Hofman thought that developmental conditions of the genital apparatus were causal in chlorosis. Forcheimer contends that intestinal auto-infection is etiological in producing the chlorotic state, since there is in chlorosis an interference with the production of hæmoglobin, the principal source of which is the gut.

Occurrence.—Chlorosis is more common in females than in males, and occurs at the time of puberty.

The condition of the blood has been described by Monti. The hæmoglobin is diminished. The number of red blood-cells is in mild cases scarcely at all reduced. In severe cases it may fall to 1,000,000 to the cubic millimetre. The absolute amount of hæmoglobin may reach 4 to 8 in 100 cubic millimetres of blood. The specific weight may be reduced to 1035. There are microcytes in the blood. There is no leucocytosis. There are poikilocytosis and polychromatophilic appearances in the stained blood.

Pseudoleukæmic Anæmia of von Jaksch.

(*Anæmia Infantum Pseudoleukæmica.*)

In 1889 von Jaksch described a symptom-complex met with among infants and young children, to which he gave the name of anæmia infantum pseudoleukæmica. He described the condition as a clinical entity which, in running its course, gives the picture of severe lymphatic anæmia. There are enormous enlargement of the spleen, slight enlargement of the liver, some enlargement of the lymph-nodes, and changes in the blood. It is a secondary anæmia rather than a distinct disease. For this reason Fischl, Epstein, and others deny that it is a clinical entity. On the other hand, Monti and Luzet have described numbers of cases. I have records of 5 cases, 1 of which was published. The anæmia is extreme.

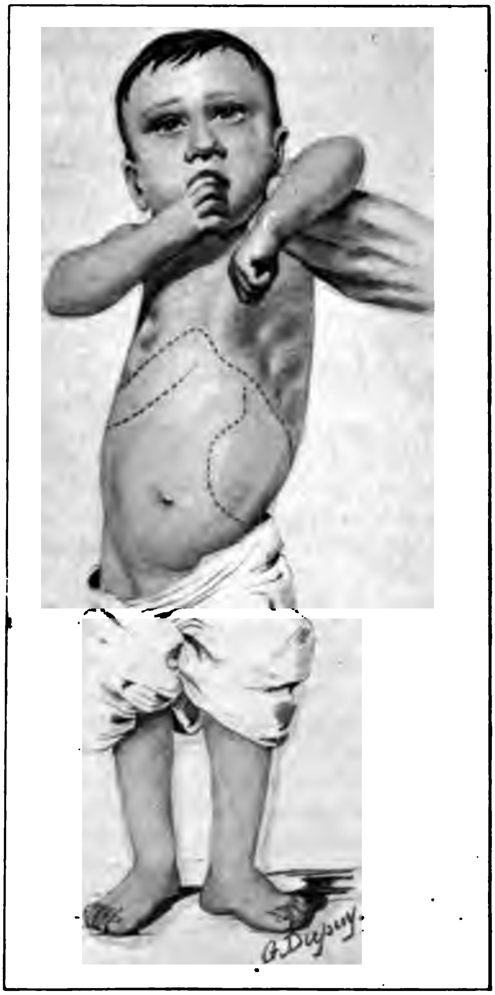
Etiology.—It is difficult to determine the etiology. Von Jaksch and Monti trace an intimate connection between this condition and rachitis. Wentworth and others regard it as secondary to some form of intestinal infection.

Occurrence.—The condition is rarely found before the age of six months. My cases ranged from the ages of twelve to eighteen months. It may occur up to the third year, and is most common from the seventh to the twelfth month. Most of the cases thus far published have occurred in infants or children suffering from rachitis or congenital syphilis. In all of my cases there were signs of rachitis. Some of the children had previously suffered from chronic gastro-enteric derangement.

Morbid Anatomy.—The spleen is large and fills the left hypochondrium, sometimes reaching the crest of the ilium. It is hard, smooth, and has a sharp border. There is an increase of the cellular elements. A small number of hæmoglobin-bearing cells of the size of red blood-cells are found.

The **liver** is slightly larger than is normal, but is of normal consistency and color. The lobuli are less distinct than is normal. Luzet has described cellular elements in the liver containing finely granular protoplasm. These are not like liver cells. They have small sharply defined nuclei, sacculated in shape. They are surmised to be forerunners of the red blood-cells. The liver cells are normal. The lymph-nodes are only moderately enlarged, and do not form lymphomata, as is the case in leukæmia. There are no changes in the bone-marrow.

FIG. 157.



Pseudoleukæmic anæmia, enlarged spleen and liver.

Symptoms.—The infants affected have as a rule suffered from chronic intestinal disturbances. Most of them are bottle-fed and atrophic. Although the skin is intensely anæmic and of a yellow, waxy tinge, there is sometimes a panniculus of fat. The musculature is flabby and the abdomen large. As a rule there are signs of rachitis. The fontanelle is open and the eruption of the teeth delayed. The infants are irritable, peevish, do not willingly take food, and do not assimilate it. In one of my cases, there was complicating pneumonia.

There is, as a rule, no fever, unless it is due to intestinal toxæmia. The picture is one of progressive emaciation and anæmia. In some cases there is complicating icterus, and the spleen reaches to the crest of the ileum. The edge of the spleen is sharp and the hilus can be distinctly felt. The liver is slightly enlarged; its edge is round and smooth. In one of my cases, it extended two and one-half inches below the free border of the ribs (Fig. 157). The lymph-nodes in the groin and axillæ are slightly enlarged, sometimes only to the size of a bean.

The Blood.—The specific gravity of the blood is reduced. The hæmoglobin may be reduced to one-quarter the normal percentage. It may be as low as 17 per cent. (Fleischl). There is a marked diminution of the number of red blood-cells. The nucleated forms of erythrocytes are abundant. There are megaloblasts, which show karyokinesis. In addition there are red blood-cells of all sizes—microcytes and megalocytes. There is poikilocytosis to a marked degree, and also polychromatophilia. The leucocytes are only moderately increased. In the severe cases the proportion of white blood-cells to the red may be as 1 : 100, 1 : 80, or 1 : 15 (Monti). The picture given by the leucocytes is different from that of leukæmia. Most authors agree that the various forms are represented and increased in equal ratio. In my cases, the mononuclear lymphocytes, as well as the transitional forms, were increased.

The **course** of the disease is chronic. In most cases, the children succumb to progressive weakness and emaciation, or to intercurrent disease. Recovery may take place. I have seen one case of aggravated form improve under treatment. Whether these cases can, as stated by Monti, pass into a leukæmia or pernicious anæmia is a matter of doubt.

Treatment.—Thus far the treatment has been empirical. Small doses of Fowler's solution are indicated. If rachitis is present, phosphorus is given by some in small doses. I have seen cases do badly under that treatment. Tonics and an easily assimilable diet are indicated. The bowels should be kept clear by enemata given daily in order to lessen the possibility of infection of the gut.

Leukæmia (Leukocythæmia).

Leukæmia is a persistent condition of the blood in which there is an increase of the white blood-cells, and a diminution of the red ones. It is a primary disease of the blood itself. Accompanying it, there are changes in the spleen, liver, bone-marrow, lymph-nodes, and lymphoid tissues. Virchow called the condition "white blood." French writers have called it leukocythæmia. The proportion between the white and the red blood-cells is not so distinguishing a feature as the appearance of large numbers of lymphocytes in the blood, in which they are normally present in only small numbers. In one form the appearance of mononuclear neutrophile-staining myelocytes which are normally absent is a distinguishing feature. Ehrlich characterizes leukæmia as a mixed leucocytosis of chronic course, since white blood-cells of all kinds are present in the blood. This is not the case in the polynuclear and eosinophile leucocytosis.

Occurrence.—The disease is rare in childhood, but some authors believe it to be more common in the first year of life than is generally supposed (Monti, Mosler). Fifteen to 20 per cent. of the cases occur in the first decade of life (Baginsky). Males are more frequently affected than females. The disease is believed to be hereditary.

The **etiology** of the affection is still unknown. In a few cases, micro-organisms and sporozoa have been found in the blood (Roux, Kelsch, Veillard, Lowit). The sporozoa of Lowit are described by him as being free in the blood as well as in the leucocytes and in the blood-making organs. In lymphatic leukæmia they are described as being intracellular only.

Some writers think that rachitis and syphilis predispose to the development of leukæmia, especially if the bones are involved as well as the liver, spleen, and lymph-nodes. Certain forms of anæmia following malaria, diphtheria, and scarlet fever, and accompanied by enlargement of the liver, spleen, and lymph-nodes, may, according to some writers, pave the way for leukæmia. Physical or mental strain, unhygienic living, defective nutrition, and traumatism of the spleen, have all been regarded as predisposing factors.

Forms.—The simplest classification of leukæmia is that based upon the anatomical appearances of the blood. Such is the classification of Ehrlich, which is as follows:

(*a*) Lymphatic leukæmia, in which there is a marked hyperplasia of lymphoid tissue.

(*b*) Myelogenous leukæmia, in which there is hyperplasia of myelogenous tissue. Lymphatic leukæmia may run an acute or a chronic course. In both forms the distinguishing feature is the appearance in the blood of large numbers of the mononuclear lymphocytes and the displacement of the polynuclear leucocytes. The acute form is rare. It occurs in childhood. Six cases have occurred in my hospital service in the past four years. Its course is rapid. There are slight or marked tumor of the spleen, slight or very marked enlargement of the liver, and a tendency to petechiæ and to general hemorrhages. Some authors regard these cases as infectious. The chronic forms show marked enlargement of the spleen.

Changes in the Blood.—As was previously stated, the lymphatic forms of leukæmia are distinguished by the appearance, in the blood, of large numbers of the small and large mononuclear lymphocytes. In the myelogenous forms, a cell which is normally not present in the blood, but is indigenous to the bone-marrow, appears in large numbers. This cell is the large mononuclear neutrophilic staining cell, the myelocyte of Ehrlich. The mast-cells are also found in these cases, but are not peculiar to this form of anæmia. In addition there is in the myelogenous forms of leukæmia an increase in the number of all three types of granulated white cells, the neutrophiles, the eosinophiles, and the mast-cells. There are dwarf forms of the white blood-cells, mitoses, and lastly large numbers of nucleated red blood-cells. Normoblasts, megaloblasts, and myelocytes are not normally present in the blood. They are occasionally found in pneumonia, and in leucocytosis. The eosinophiles are increased to fifteen times their normal number. The slow coagulability of leukæmic blood is characteristic.

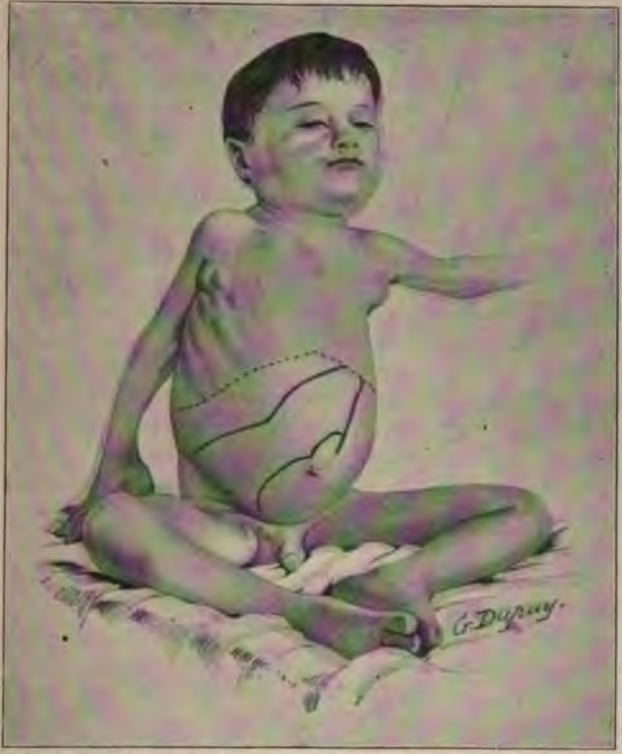
The spleen is enlarged. It is at first soft, often firm, and is infiltrated with lymph-cells. The capsule is thickened; the connective-tissue stroma is increased and infiltrated with lymph-cells. The lymph-nodes show similar changes, and may be enlarged, forming tumors of considerable size.

The bone-marrow is so infiltrated with lymph-cells as to acquire the appearance of a purulent infiltration. The same lymphoid infiltration is found in the liver. The follicles of the gut are swollen. There is an increase of lymphoid cells and tissues. The lymphoid tissues elsewhere, such as the tonsils, thymus, skin, and even the retina, show the same changes. There are hemorrhages and exudate in the ear, and the nerves and nervous tissue of the central nervous system are the seat of lymphoid cellular invasion.

Symptoms.—**Acute Leukæmia.**—Cases of acute leukæmia in infancy and childhood have lately been increasing in the literature. The most recent cases include those of McCrae, in a boy aged three years, and of Miller, in an infant of eight months. Cases have also been reported by Morse, Japha, Strauss, Monti, Berggrün. The symptoms in all the published cases were similar. In a boy eight years old, admitted to my hospital service, there were no premonitory symptoms. Two months before admission he was in good health.

He became very pale, there were irritability and loss of appetite, and the abdomen increased markedly in size. He complained of pains in the legs, and at the onset had chills and fever every other day. After the appearance of the chills he suffered from a low irregular fever. A week before death, the skin had a waxy color, there were petechiæ on the extremities, the gums bled easily, and the lymph-nodes of the axillæ and groin were enlarged. There was an anæmic murmur with the first sound of the heart; the liver was enlarged below the free border of the ribs to the extent of two fingers' breadth; the spleen was enlarged to the level of the umbilicus; the fundus of the eye showed retinal hemorrhages. Examination of the blood showed the hæmoglobin to be reduced to 15 per cent. (Fleischl). The red blood-cells numbered 1,012,000 to the cubic millimetre; the white blood-cells, 37,000. There was an immense preponderance of lymphocytes (mononuclear). The patient died with signs of progressive weakness. Coma was preceded by vomiting

FIG. 158.



Acute lymphatic leukæmia. Enlarged lymph-nodes, spleen, and liver. Boy four and one-half years of age.

and the appearance of a few petechiæ. The blood state continued much the same as at first. In another case the number of mononuclear lymphocytes was fully 75 per cent. of the white blood-cells. In both these cases the spleen and liver diminished before death. The proportion of white to red blood-cells may not be far from normal. In another case the nucleated red blood-cells, large and small, were very numerous. In this case, in a boy of four and one-half years, the nodes around the parotid and angle of the jaw, in the axilla, and in the inguinal region, increased in a short time to a large size, and the spleen grew rapidly larger and reached to the crest of the ilium. The liver reached to the umbilicus. These mediastinal lymph-nodes were enlarged and caused great dyspnœa. The distress was very great just before the lethal issue (Fig. 158). In a case of v. Noorden's the proportion of the white to the red blood-cells was 1 : 200. The predominance of the lymphocytes is diagnostic. Most of the cases published showed a slight temperature. The fatal issue usually results a few weeks or a month or two after the onset of symptoms.

The Chronic Form.—The symptoms of the chronic form extend over a greater length of time. For months there are anæmia, lassitude, and extreme physical weakness. The appetite is good, but in spite of abundant nourishment, emaciation is progressive. In some cases there are periodic diarrhœal attacks. Profuse hemorrhage may occur without warning either from the nose or intestines. Chills and fever resembling those of paludism are sometimes present. None of these symptoms is particularly characteristic. As the disease progresses there are headache and pain in the limbs and in the region of the spleen. The anæmia after a time assumes a severe type, and the skin becomes waxy and yellow. At this stage the spleen and liver enlarge and distend the abdomen. There are dyspnœa and palpitation; the anæmia takes the hydræmic form, and there is œdema of the face, hands, and feet. Hemorrhages then occur from the nose, lungs, mouth, intestines, but rarely from the kidneys. There are petechiæ in the skin and hemorrhages in the retina. In the lymphatic form the lymph-nodes in various parts of the body enlarge and form masses which are painless and covered with unaffected skin. The skin may be affected by the process. The mesenteric nodes may sometimes be felt through the abdomen. The spleen attains an enormous size. The liver may extend as far as the umbilicus. Respiratory difficulties, heart weakness, and nervous symptoms (such as vertigo, somnolence, and coma) end the clinical course of the disease. The urine is diminished, and contains hyaline casts, lymphoid cells, and a trace of albumin. There may be a slight continued fever in the course of the disease.

The **prognosis** is very unfavorable. Of 39 cases collected by Birch-Hirschfeld, only 4 recovered. Only in the early stage is recovery possible. Death supervenes from exhaustion with hemorrhages or from intercurrent pleuritis or pericarditis.

The **treatment** of a disease whose exact nature is still unknown is difficult. Good food, and hygienic surroundings are the first requisite. In the treatment of anæmia, the iodide of iron, cod-liver oil, and arsenic are the chief drugs employed. In the lymphatic form, arsenic in the form of Fowler's solution gives the best results.

Hodgkins' Disease.

(*Anœmia Lymphatica; Adénie; Pseudoleukœmia; Lymphadenoma.*)

This disease is really not an affection of infancy and childhood inasmuch as 75 per cent. of the cases occur above the age of ten years. It is mentioned here to emphasize its characteristics as distinct from tuberculous adenitis or scrofulous enlargements of the lymph-nodes. It is an affection beginning with the enlargement of the lymph-nodes of the neck, usually of one side, and accompanied by an enlarged spleen and liver. In the spleen, liver, and other organs there are nodular growths. There is a progressive cachexia accompanied by febrile periods. The disease is fatal either in a short time of a few months or after a period of two or three years, during which there may be intervals of improvement. There are no changes in the blood such as are seen in true leukæmia, and in this lies the main element of differential diagnosis. A most complete account of the nature of this rare affection will be found in a recent monograph by Dr. Reed, published in the Johns Hopkins Hospital Reports, Vol. X., and in a monograph by Clarke in which he collected 43 cases.

The Hemorrhagic Diatheses.

In this class of diseases are embraced only those affections which are due to some primary change in the blood or in the circulatory apparatus. Thus conditions which are due to local disease of some organ, or the hemorrhages which follow the acute infectious diseases or drug poisoning are not included. Experimental pathology has as yet not given any clue to the etiology of the hemorrhagic diatheses. The contention of William Koch and Ajello, that they are infectious diseases or are due to some auto-intoxication, is not universally accepted. At present the clinical classification of these diatheses into the transitory forms in which are included purpura simplex, peliosis rheumatica hæmorrhagica, scorbutus, and the persistent form hereditary in character, such as hæmophilia, may be accepted. In the latter, the hemorrhage may be extensive, difficult to control, and due to some very slight cause.

Simple Purpura.

This is a transitory condition characterized by small hemorrhages or petechiæ, or large, irregularly shaped extravasations of blood. These are as a rule discrete, but may be confluent, and are situated in the epidermis or in the superficial layers of the cutis. Immediately after the extravasation they have a bluish-purple tinge. After a few days they become brown or greenish yellow. These extravasations are seen most frequently on the lower extremities, generally on the extensor surface. They also occur in other localities. As a rule there are few or no symptoms. There may be crops of petechiæ appearing at short intervals. In a few cases there are, after an exacerbation of the local phenomena, loss of appetite, vomiting, and general malaise. The so-called purpura cachecticorum appears on the body, abdomen, back, and upper extremities in children under two years, suffering from diarrhœa and other exhausting diseases. In the latter case there may be leucocytosis, due to the original affection. The changes in the blood in simple purpura are still to be studied.

Etiology.—The cause of this purpura is still unknown. It may be due to some obscure toxæmia.

The **prognosis** is very good in the primary form. In the secondary form it will depend on the nature of the original affection.

The **treatment** will depend on the nature of the original disease. I treat the purpura itself in the same manner as cases of purpura hæmorrhagica, which will later be fully described.

Hæmophilia.

Hæmophilia is a rare condition which may be congenital or hereditary. It becomes apparent at birth or in early infancy, and is rare in later life.

The **nature** of the affection is obscure. It is a type of hemorrhagic diathesis which is transmitted from generation to generation in the female line. It is characterized by the occurrence of uncontrollable hemorrhage after very slight injuries, and operations, and also in the absence of known traumatism.

Etiology.—Many theories of the cause of the affection have been advanced. They may be grouped as follows:

(*a*) An abnormal delicacy and friability of the bloodvessels.

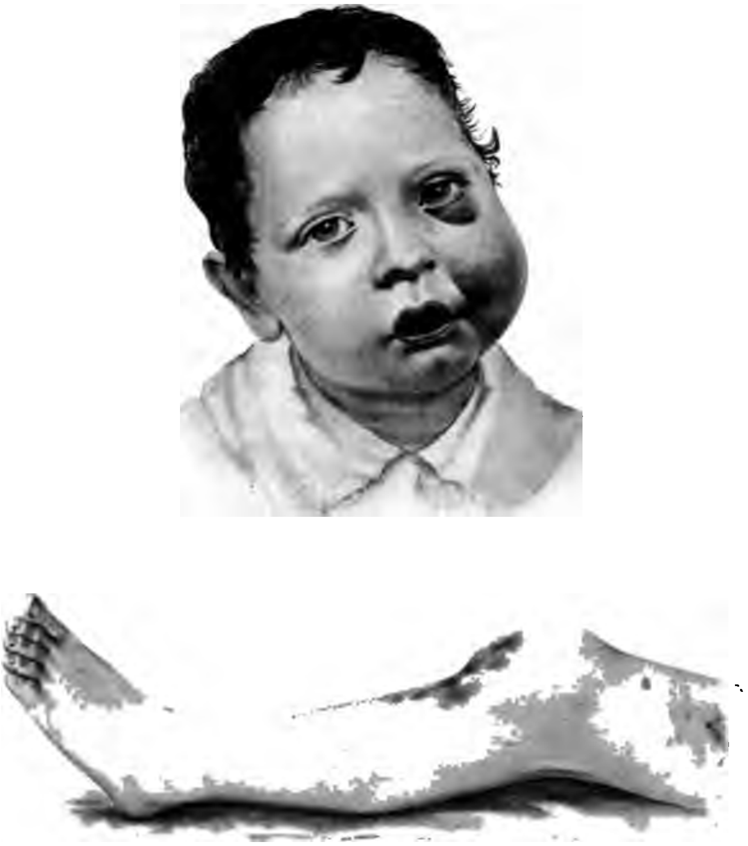
(*b*) An increase of the volume of blood (Immerman).

(*c*) A defect in the coagulable constituents of the blood.

(*d*) Certain agencies acting as toxins on the elements of the blood, causing their dissolution (Koch).

The condition is most common in the Slavic races. Children dying of the affection show evidences of intense anæmia, but may be well nourished. Virchow has demonstrated that there is a narrowness in the arteries and also a thinness of their walls. Birch-

PLATE XXV.



Hæmophilia. Boy six years of age. Hæmatoma of the face; hemorrhage into the knee-joint. (Case of Dr. Martin Ware.)

Hirschfeld found that the endothelium of the arteries was enlarged, and that the nuclei were swollen. The blood itself shows no changes except those proper to post-hemorrhagic anæmia. The hemorrhages may occur in any region and from any organ of the body. There may be hemorrhage into joints, profuse epistaxis, intestinal hemorrhage or uncontrollable hemorrhage from the mouth or lung. The drawing of a tooth, the incision of an abscess, or a minor operation such as circumcision, may cause uncontrollable and fatal hemorrhage. In the newly born infant, there may be fatal hemorrhage from the cord. In the case pictured in Plate XXV. there were hemorrhages into the joints and into the face, without distinct traumatism. This case came of a family of bleeders in which there had been fatalities following circumcision.

The condition lasts weeks, months, or years—in fact, it persists during the life of the individual. Some authors believe that the female members of families thus affected should not marry.

The **treatment** is mainly prophylactic. The infant should nurse a wet-nurse, in order that the noxious influence of its own mother's milk may be lessened. Good food and fruits of all kinds should be given. All operations and traumatism should be carefully avoided.

Purpura Hæmorrhagica.

(*Morbus Maculosus Werlhofii.*)

In the prodromal period before the appearance of the hemorrhages, there may be several days of general malaise, disturbance of appetite and digestion, and febrile movement. There are anæmia, pain in the limbs, and œdema of the feet. The hemorrhages may appear without any symptoms. They are especially frequent in the lower extremities, and next most frequent in the upper extremities and on the chest, face, and trunk. They consist of extravasations of blood in the skin and subcutaneous tissue. The mucous membranes are frequently affected. Epistaxis, bleeding of the gums, bloody movements, and bloody urine result. There are ecchymoses in the conjunctiva and bleeding from the ear. The hemorrhages in the skin may be petechiæ, or irregular bluish or purple blotches which subsequently become yellowish or greenish yellow. They occur spontaneously or follow slight traumatism or pressure. There may be hemorrhages into the joints. There may be exacerbations and recurrences of hemorrhages extending over weeks. The tendency of the mucous membrane to bleed has been mentioned. The gums are spongy and bleed easily. There are hemorrhages or petechiæ on the soft and the hard palate. The hemorrhages from the kidney cause the appearance of albumin and blood in the urine. The urine is red and blood-coloring matter may be found by the turpentine-guaiac test. Hemorrhages in the brain and central nervous system may occur, causing paralyses and coma. In mild cases there is no disturbance of nutrition, but in severe ones the uræmia is marked, as is also the emaciation. The blood shows few changes. The number of red blood-cells is diminished, as is also the specific gravity. In severe cases there is a slight leucocytosis; the polynuclear leucocytes are increased, eosinophiles are few, microcytes are present, and there are a few normoblasts. The leucocytosis improves as recovery sets in.

Course.—The cases of ordinary severity recover. Severe cases may recover or may result fatally.

The **etiology** of this affection is still obscure. Because of its infectious nature, William Koch believes it to be allied to scorbutus and other hemorrhagic affections. His view is not supported by other writers. Ajello and Schwab regard the condition as an auto-infection or a form of toxæmia. Kolb, Tizzoni, and Babes have found bacteria in the blood of fatal cases. Others have isolated streptococci and staphylococci from the blood (Lebreton). In one of my cases there was a history of an insect-bite. The disease is rare in breast-fed infants and is more common after the age of two years than before. The infants and children attacked may have previously been in good health.

The **diagnosis** is made from the course of the affection and the size and nature of the hemorrhages. The constitutional disturbance is more marked than in simple purpura. The hemorrhages are blotches, in that respect differing from the petechiæ of peliosis. The joints are not swollen, as in the latter affection.

The **treatment** consists in placing the patient in hygienic surroundings, and giving a nutritious diet with a liberal allowance of fruit and vegetable acids. In marked cases, Fowler's solution, given in moderate doses, gives good results.

Purpura Rheumatica.

(*Peliosis Rheumatica of Schönlein.*)

Purpura rheumatica consists of an eruption of small discrete purpuric spots in the vicinity of the large joints of the extremities, and occurring especially on the lower extremities above the knee. The accompanying symptoms are pain and swelling of the joints of the lower or upper extremities.

Symptoms.—Slight fever is followed by the appearance of the purpuric spots and the swelling of the joints of the lower and rarely of the upper extremities. The joints are painful, as in rheumatism. At times the swelling of the joints is less apparent, but there is nevertheless tenderness on pressure. The purpuric spots are particularly numerous in the vicinity of the joints. A general urticaria may precede the appearance of the purpura. There are no heart complications. The condition of the blood is not as yet understood. There may be several crops of purpuric spots appearing at intervals of days or weeks. In other cases there are œdema of the face and enlargement of the spleen. In one of my cases there were at first slight hemorrhages from the bowel. There may be epigastric pain and tenderness in the course of the disease.

The average duration of the affection is from ten to fourteen days. There may be relapses extending over weeks.

The **etiology** is obscure. The disease occurs in children previously healthy. It is seen in older children only, and has no apparent relation to acute articular rheumatism.

The **prognosis** is good even when there are several relapses and when the affection takes a subacute course.

Treatment.—Rest in bed is the first requisite of treatment. A nutritious diet in which there is an abundant allowance of fruit and vegetable acids is given. Lemonade and orange-juice are especially indicated. The bowels are regulated with the salicylate of sodium given in moderate doses. A child four years of age is given grains v (0.3) three times daily. The pains in the joints are easily controlled by rest. In the subacute stage small doses of Fowler's solution are of great benefit.

Henoch's Purpura.

Henoch in 1874 described a series of 4 cases of purpura which he classified as distinct from purpura hæmorrhagica or peliosis rheumatica. The symptoms were as follows: Children apparently in good health were attacked by a form of purpura in which there were arthritic pain, vomiting, and intense abdominal pains with bloody diarrhœa. The rheumatoid pains were accompanied by swelling of the joints. The purpura was of the hemorrhagic type—that is to say, there were extravasations of blood in the form of ecchymoses or raised exanthematic areas, not disappearing on pressure. The areas were situated on the abdomen and lower extremities. The joints affected were those of the wrist, elbow, and ankle. The intestinal symptoms consisted of repeated vomiting, tympanites, excruciating colicky pains, bloody stools, and tenesmus. One case was fatal. Such cases have been from time to time described by other observers. I have seen a number of cases. These cases are at present regarded as due to a form of intestinal infection the exact nature of which is still obscure. They constitute a group probably belonging to the class of primary hemorrhagic affections in which is included the so-called peliosis rheumatica.

Diagnosis of Forms of Purpura.—It is not always possible, clinically, to assign each form of purpura to its proper class. This is especially true with young children, in whom there occur forms of purpura showing a diversity of symptoms and not fitting into any sharply defined class. Nor is it always possible at the bedside to decide whether the condition present is scorbutus or idiopathic purpura. Characteristic of both purpura and scorbutus are the hemorrhages into the skin, the internal organs, the serous cavities, and the mucous membranes. On the other hand, the frequency of hemorrhages and affections of the gums, the prodromal cachexia, the joint-affections, and the periosteal hemorrhages are peculiarly characteristic of that form of scurvy called Barlow's disease, which is seen in nurslings and young children. The purpuric affections of so-called idiopathic type, in which a purpuric exanthema is spread over the whole surface, may be called simple purpura.

In the so-called rheumatic purpura or peliosis rheumatica there is a blotchy hemorrhagic exudate over the surface in the vicinity of the joints, with pain in the joints, and gastric pains. There is always a tendency to relapses. Hemorrhages from the mucous membranes and bowels are rare, but occasionally occur.

In purpura hæmorrhagica or morbus Werlhofii there are minute or blotchy hemorrhages in the skin and internal hemorrhages from the mucous membranes, stomach, and intestines. Attempts to define sharply each of these sets of cases have been made. It is not always possible or desirable to do so. I have seen cases of peliosis with bowel hemorrhages and gastric crises, and cases of purpura hæmorrhagica in infants, in which there were pains in the joints, evinced by the distress shown when the joints were moved. The forms of purpura regarded by Henoch as a distinct type are classed by others as purpura rheumatica. The different classes of idiopathic purpura therefore overlap, one case frequently showing symptoms of two types. The only possible conclusion is that there may be a common cause of all forms of purpura—probably an infection.

Pernicious Anæmia.

This is a primary anæmia which causes progressive impoverishment of the blood and results in death. It is not common in infancy and childhood. The condition of the blood in infancy and childhood has not as yet been closely studied. The changes in the blood which have been published as characteristic of this condition in infancy and childhood are found in other states, such as the severe anæmia of rachitis and syphilis. Ehrlich is not disposed to accept these cases without question. Blood pictures which in the adult may be diagnostic of pernicious anæmia cannot be thus interpreted when found in infants and young children. Observers of note, such as Monti, Berggrün, and Baginsky, have published cases in infants and young children. I have met a case in an infant which had been bitten by a rat. After an interval, anæmia of a progressive and fatal type set in. The changes in the blood were similar to those characteristic of the same form of anæmia in the adult. Monti has collected 16 cases, 2 of which were in infants; 5 ranged from one to six years; 9 were above the age of five years. On the other hand, Ehrlich found that of 240 authentic cases, only 1 occurred in the first decade of life. That case was in a girl of eight years. In the face of such great diversity of opinion, it is wise to await the results of further research. For the purpose of reference, the following account of the changes in the blood which, according to Ehrlich, are diagnostic of pernicious anæmia in the adult, is appended:

(*a*) The volume of blood is markedly diminished.

(*b*) The color is at first normal, but later resembles that of beef-water.

(*c*) The hæmoglobin may be as low as 10 per cent. (Fleischl). This is due to a diminution of the number of red blood-cells, for the individual cell may have a hæmoglobin content equal to the normal or above it.

(*d*) There are microcytes, megalocytes, and sometimes gigantocytes. The megalocytes may constitute 70 per cent. of the red blood-cells. They become fewer on convalescence. There are few megaloblasts, but characteristic normoblasts are found.

(*e*) Clumps of free granules are found in the blood. The red blood-cells may contain granules.

(*f*) Staining solutions produce polychromatophilic effects.

(*g*) The eosinophiles are normal in number.

(*h*) The number of white blood-cells is diminished as well as that of the polynuclear neutrophiles. The latter condition indicates serious involvement of the bone-marrow. The lymphocytes are proportionately increased.

(*i*) The leucocytes show no changes. Improvement is ushered in by leucocytosis.

(*j*) The specific gravity of the blood is diminished, as is also its coagulability.

In my case the nucleated red blood-cells were numerous.

Infantile Scorbutus or Scurvy (BARLOW).

(*Acute Rachitis* (MÖLLER); *Barlow's Disease, Hemorrhagic Rachitis* (FÜRST); *Scurvy Rickets* (CHEADLE); *Hemorrhagic Periostitis* (SMITH).

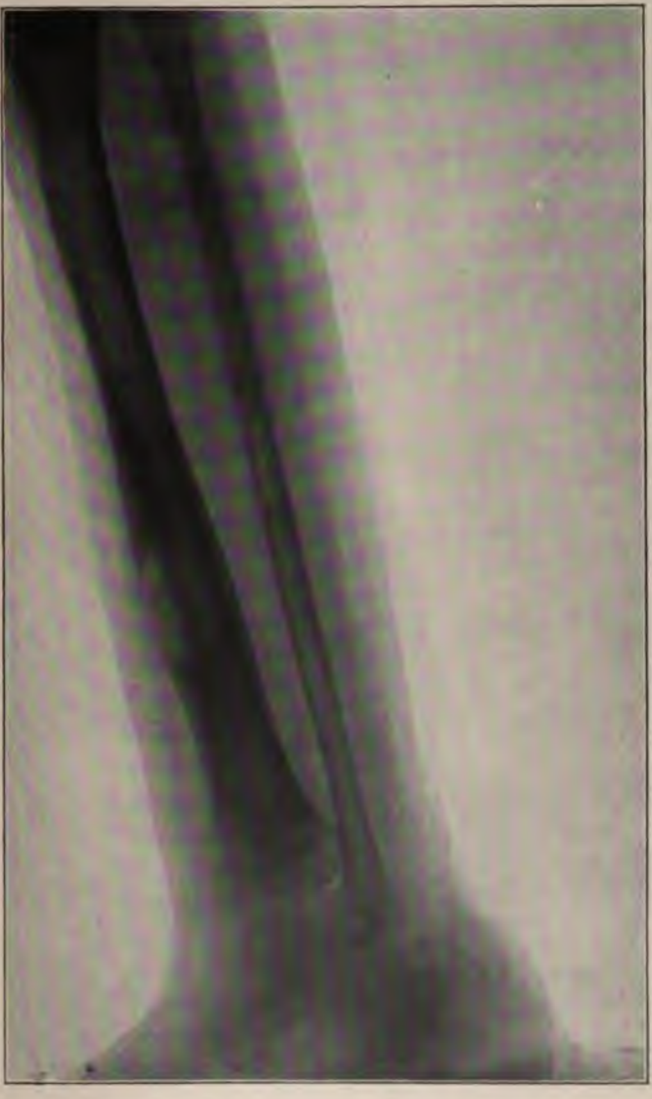
History.—Cases of this affection are described in the literature under the name Acute Rachitis, which was given by Möller, 1859–1862. The first definite clinical description of the disease under its present title was made by Barlow, Cheadle, Gee, and others of the English school, completed its clinical study. Northrup and Crandall have made it familiar to American physicians.

Occurrence.—The disease occurs chiefly in infants and in children under the age of two years. Under certain conditions it also occurs in older children and in adults. The majority of the 372 cases collected by the committee of the American Pediatric Society, occurred between the sixth and fourteenth months. The ninth month showed the greatest percentage of the cases occurring before the end of the second year. The sexes were equally affected. A second attack was recorded in a case of Holt's. In a case which I saw recently, there were two attacks.

The Nature of the Affection.—The nature of scurvy as it is seen in infants and children is still obscure. It is undoubtedly a form of hemorrhagic diathesis, which attacks subjects susceptible because of previous abnormal constitutional conditions and defective nutrition. There are several theories as to its exact nature. None is universally accepted. Some insist that it is a form of acute rachitis (Möller, Förster, Böhm, Steiner, Fürst, Ausset). Others contend that it is a form of scorbutus (Barlow, Northrup, Crandall, Netter, Rehn, Pott). Some of the English school regard it as a combination of scurvy and rickets (Cheadle, Gee, West). To the latter contention Heubner, Schoedel, and Nauwerck give most support. These authors insist that the disease supervenes only in an organism already affected by slight or marked rachitis. On the other hand, there are authors who, like Schmorl and Naegeli, think that the affection is *sui generis*. Some authors have endeavored to establish a correlation with congenital syphilis. The consensus of clinical opinion, however, tends toward the acceptance of the theory of the scorbutic nature of the affection.

Etiology.—The essential exciting cause is not yet known. The theory of the toxæmic or infectious nature of the disease has been advocated by William Koch. Bacteria of various kinds have been found in the blood, but there is little uniformity in the results of studies. In all the cases thus far studied the nature of the diet, breast-milk, raw cows' milk, sterilized or pasteurized milk, or some artificial food, has been a strong predisposing factor. The diet has been insufficient for the nutrition of the patient, but what special element has been lacking in the food is still obscure. In the collected results of the investigations of the American Pediatric Society 10 infants were wholly breast-fed; 2 were partially breast-fed; 4 took raw milk. The greater number, 68, were brought up exclusively on sterilized milk; 16 took pasteurized milk. The others took foods of different kinds. It may be that the mode of preparing the food (raw cows' milk, pasteurized or sterilized milk) is of less importance in paving the way for the onset of this affection than its inherent composition. Cases have been cured in part by changing the

PLATE XXVI.



X-ray of the Bones of the Leg in a Case of Scorbutus, showing the hemorrhage under and in the periosteum of the tibia at the junction of the middle and lower third of the bone.

composition of the food, also by substituting sterilized for pasteurized food, and *vice versa*. The very fact that breast-milk has been the exclusive article of diet in some cases should direct attention to the fact that the affection may be caused by lack of some necessary element in the diet. This view is commonly taken at present. It is interesting in this connection to consider the contention of the celebrated Arctic explorer Nansen, that if exercise and fresh air are taken, and abstinence from alcohol is maintained, scurvy on voyages will be unknown if foods are carefully sterilized and devoid of toxins and ptomains. The latter, he insists, exist in most of the milk, fish, and food eaten on voyages. Although in the most aggravated cases of scurvy that have come under my notice the diet has been sterilized milk, many infants who take that food prepared properly do not develop the disease. Some authors believe that the success of antiscorbutic treatment with vegetable acids indicates that the organism has been for a time deprived of some essential food element. In the presence of a concrete case attention should first be directed to securing fresh food of proper composition.

Rachitis.—Much has been said as to the connection of rachitis with this disease. The investigations above referred to show that fully 45 per cent. of the cases occurred in infants and children who showed clinically signs of rachitis. This does not account for cases in which rachitis may exist, but may not be apparent except on microscopic examination (Hirschsprung, Schoedel). The majority of cases examined post mortem showed the changes of rachitis (Schoedel, Schmorl).

The **morbid anatomy** has been carefully and extensively studied by Schoedel, Nauwerck, and Schmorl, whose results agree in all essentials.

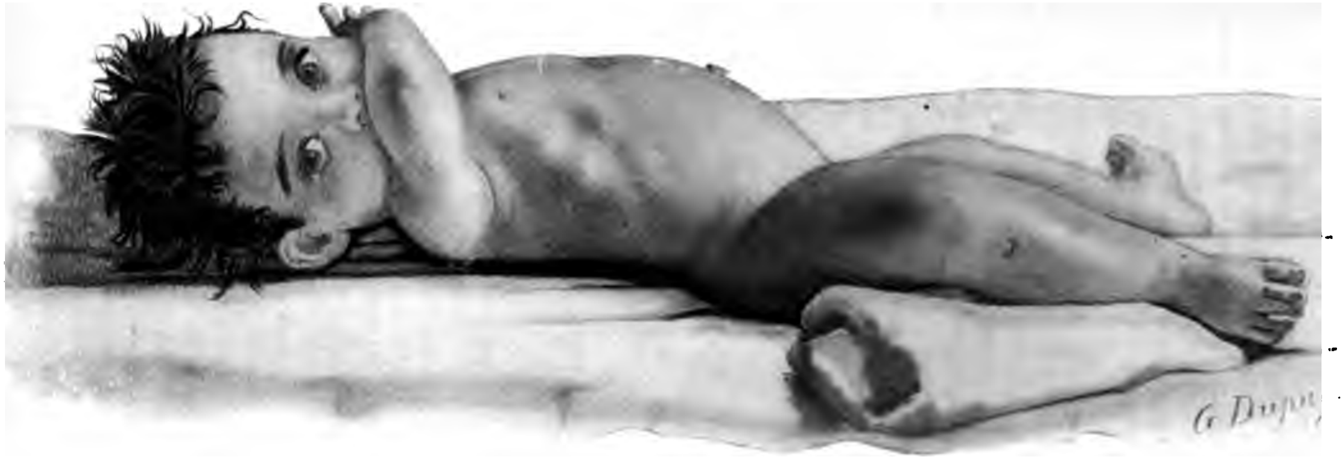
The **bones** in most cases show the changes seen in rachitis. There are disturbances of growth and of bone formation. There is an increase in the width and vascularization of the cartilage zone. There are irregularity of the calcification zone, and a pathological formation of osteoid tissue. The changes at the epiphyseal junction and the periosteum are those seen in rachitis. The ribs are the bones most frequently affected, the next greatest frequency being in the bones of the lower and upper extremities. The changes caused by scurvy consist of hemorrhages into the loose vascular layer of connective tissue of the periosteum adjacent to the bone. Thus the hemorrhages are intraperiosteal and subperiosteal (Plate XXVI.). They may be of considerable extent, either in the vicinity of the epiphyseal junction or in course of the shaft of the bone. They may form a layer several millimetres or centimetres in thickness. The outer layer of the periosteum, the fibrillar connective-tissue strata, is not the seat of hemorrhage except in the severest cases. The layer of periosteum next the bone is thickened. The hemorrhages are both recent and old. Hemorrhages of both kinds are found in the medullary canal. The morbid changes are most marked in the ribs, next in the femur and in the bones of the upper extremities. Some of the long bones show loosening and even separation of the epiphyses and diaphyses. The infractures or fractures are of this nature. The fragments may override. In such cases the hemorrhage is great. The marrow of the bones loses its lymphoid character and becomes gelatinous.

There are subpleural and subepicardial hemorrhages. The spleen is enlarged, owing to the presence of rachitis. Slight subcutaneous hemorrhages may extend into the muscular tissue. There are hemorrhages into the mucous membrane of the hard palate and gums.

Symptoms.—Mild cases sometimes escape notice. An anæmic infant may cry when bathed or may favor one extremity. It may hold one thigh rigid or cry when the limb is handled in the process of diapering. Mothers at first suspect traumatism. The infant develops slight ecchymoses on the tibiæ, and is then brought to the physician. If there are teeth, there may at this stage be no swelling of the gums or of the extremities. There is no fever; there may not be any anæmia. In the severer cases the symptoms are more marked. The skin in the infant of from seven to nine months of age acquires a pallid or greenish tinge. The infant cries when touched. One or both of the lower extremities lies as if paralyzed. If an attempt is made to move them, the infant appears to feel pain. The limb is swollen in the course of the shaft or in the vicinity of the knee or ankle, the swelling extending up the shaft (Plate XXVII.). The ribs are apparently tender. There may be one or two subcutaneous ecchymoses on the surface of the body. If there are teeth, the gums, especially those of the upper jaw, are swollen into cushion-like formations. These bleed easily and may partly conceal the teeth. If there are no teeth, the gums may appear normal, or the free border, especially of those of the upper jaw, may have a bluish, swollen appearance, which may be very slight or quite marked. The infants may have a capricious appetite, may take little of the bottle or may nurse ravenously.

The very severe cases have, as a rule, been allowed to run on for months in the belief that the infants were suffering either from rheumatism or dropsy. For some time before coming under treatment, the infant has cried when diapered or when the shoes or stockings were put on; later it becomes pale and loses ground. The appetite is poor. The thighs and the ankles begin to swell. The child does not move the extremities, which are swollen to twice or three times the original circumference. Ecchymoses appear on the surface of the swellings of the legs and thighs. Parts of the skin acquire a bluish-green, bruised appearance. Deformity occurs in the thigh, especially at the junction of the diaphysis with the head of the bones.

PLATE XXVII.



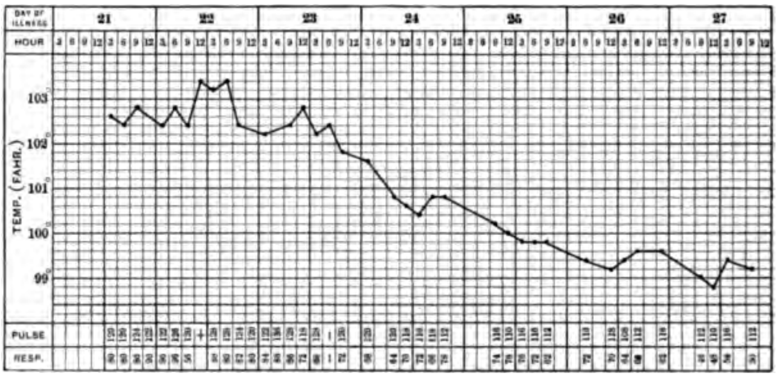
Scorbutus in an Infant aged Seven Months who was Fed on Sterilized Milk. Great swelling of the right thigh and leg, due to intraperiosteal hemorrhage. Swelling of the right knee and ankle. Ecchymoses over the greater portion of the thigh, leg, and dorsum of the foot. The left thigh and leg involved to a lesser degree. Complete disability of the lower extremities, especially the right, with great pain on manipulation. Swelling of the costo-chondral junction of all the ribs. Suggillation of the left eye. Complete and uncomplicated recovery.

This is due to infracture or loosening of the epiphyses at the epiphyseal line. The costochondral junction of the ribs is much swollen. There is a distinct series of very large swellings in this locality which are due to hemorrhage into the line of juncture of the rib and cartilage. Ecchymoses and sugillation appear about the orbit. The face and eyes have an œdematous, hydræmic appearance. The gums may not be at all affected, but if the infant has teeth there may be sponginess.

When the physician examines the infant, he finds that the pain produced by the procedure causes it to shriek with agony. The ribs are painful to the touch. The swellings on the thigh are uniformly fusiform, and, as a rule, hard and not fluctuating. The abdomen is tense and tympanitic. The infant has had some bleeding from the nose, but not necessarily from the bowel. In other cases there are not only hemorrhages from the bowels, but also from the kidney, in the form of hæmaturia. There may be albumin and casts in the urine, or these may be absent.

Of especial interest are those cases in which hæmaturia is the chief and only objective symptom of the disease. Such cases as I have seen the infants may be in excellent physical condition, of good weight and color, and still for a period of weeks have voided urine which contains blood, but no casts. Careful examination of such cases will reveal a tenderness of the tibiæ, or a just perceivable swelling of the gums or a very narrow blue line along the gums. I have recently seen a number of cases of scorbutus in which the main symptom was the appearance of blood in the stools, simulating a dysentery. In another case, that of a child twenty-two months of age, the first symptom of scorbutus was a sharp hemorrhage from the bowel. This hemorrhage was repeated, but was not as profuse as the initial

FIG. 159.



Temperature-curve of a case of scorbutus in an infant seven months of age. Resorption fever. The chart shows the very high number of respirations as compared to the pulse. Cause of high respirations probably pain and extreme anæmia. The curve taken from the start of treatment.

one. A careful examination in this case revealed a slight tenderness of the tibiæ and a tendency to ecchymoses following the least traumatism.

The pulse is, as a rule, not increased. In one case without complicating pneumonia, in which I found the respirations enormously increased, I reached the conclusion that the increase was due to the pain and extreme anæmia.

In severe cases there may be slight temperature (Fig. 159), which may be due to resorptive fever caused by the immense extravasations of blood.

The hemorrhages in the skin may be localized in the form of minute petechiæ or there may be ecchymotic blotches of considerable size. The latter may appear over the swellings along the bones.

The fractures or infractions were present in only 9 cases of the set collected by the American Pediatric Society. The gums were generally affected in infants with teeth, and were swollen and spongy in 24 cases in which there were no teeth. They may be normal in severe cases if there are no teeth, and swollen in mild ones. The symptoms in older children resemble those of adults. In one case in a child over two years of age the surgeons of a dental clinic had been consulted for an uncontrollable bleeding of the gums. The child had ceased to walk on account of pains in the lower extremities, which had been interpreted as rheumatic. In older children the gums are affected, and the hemorrhages take the form of petechiæ and blotches, appearing in crops over the surface of the body as in the adult. They have joint-pains and malaise.

Prognosis.—The disease in infants and children gives a very good prognosis if recognized and treated in time. Most cases recover. The fatal cases are those in institutions or elsewhere in which the diagnosis has not been made or in which death has been caused by some intercurrent affection, such as cerebral hemorrhage, diarrhœa, or pneumonia. In 379 cases collected by the American Pediatric Society the mortality was 8 per cent.

Duration.—There is no fixed duration. Much depends on an early diagnosis. Even if the disease has existed months before a diagnosis is made, the patient may still recover. The great danger is that a hemorrhage may occur in the cerebrum or that the infant may contract an intercurrent affection through exhaustion. If allowed to continue without treatment, the disease may cause exhausting intestinal hemorrhages or hemorrhage of great extent elsewhere, with consequent anæmia and death.

The **diagnosis** of infantile scurvy presents no difficulties. The pains in the extremities, the paralytic phenomena, the swelling of the gums, the swelling in the vicinity of the joints of the limbs or along the shafts of the bones, the swellings on the ribs, and the ecchymoses in the skin and about the eye, are all characteristic. The pareses of the upper extremity are frequently mistaken for those due to syphilis. The history, and the absence of syphilitic eruptions will aid in diagnosis. In the presence of a hæmaturia in an artificially-fed infant, where other causal elements fail we should always think of the possibility of scurvy. In cases of prolonged enteric catarrh, in which the infants are emaciated and pass pure blood with the movements, scurvy should be thought of. I have seen a case of scurvy with hemorrhages from the bowel mistaken for intussusception, and operated with this mistaken diagnosis in view.

The **treatment** of infantile scurvy is simple and satisfactory. The infant is given fresh, pure milk properly modified. The milk should be given raw, and in summer should be kept well packed in ice. In addition, orange-juice and lemonade are given in the course of the day. An infant seven months old should have 2 ounces of lemonade and ½ ounce of orange-juice in twenty-four hours, given every two hours after each nursing. Some authors advise the giving of raw beef-juice, but it is not necessary. After two weeks the quantity of fruit juice should be reduced, but a small quantity of orange-juice should be given daily for some time. Medicines are not indicated except for the anæmia, which is best treated by doses of half a drop of Fowler's solution given three times daily, or by some easily assimilable peptonate of iron.

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SECTION X.

DISEASES OF THE BONES.

General Facts.—In examining the joints, it should be borne in mind that the bones entering into the formation of the joints may be affected. The diaphysis may be diseased without accompanying involvement of the joint.

Tuberculosis.—In all bone lesions tuberculosis should be excluded. In infants and children, the question as to whether the existing condition is tuberculosis of the bone or syphilis is constantly arising.

Syphilis affects by predilection the long bones in the diaphysis, while tubercle affects the short bones, especially in the vicinity of the joints. In this region, also, tubercle attacks the epiphyses of the bone and may thus involve the joints secondarily.

Pain in syphilitic bone lesions is very marked, acute, and with nocturnal exacerbations; while the pain of tubercular bone lesions is obscure and indefinite, although persistent.

The swelling in syphilis is in the form of a periostitis or an ostitis involving only the bone; in tuberculosis, the surrounding tissues are affected as well as the bone, and abscess and fungous granulation result.

Syphilis rarely suppurates; the contrary is true of tuberculosis.

Syphilis of the bones does not as a rule lead to cachexia; tuberculosis of the bone eventually causes cachexia and emaciation.

There are cases in which doubt will arise as to the true nature of the bone affection. This is especially the case when the small bones of the hand are affected.

Sudden painful swelling of the long bones occurring in corresponding bones on both sides should awaken a suspicion of syphilis, even in the absence of other signs of syphilitic disease. A long bone which has been affected by syphilis will be irregularly thickened, owing to the repeated attacks of periostitis. This thickening is likely to be confounded with that caused by rachitis.

In rachitis, the bone is less painful than in syphilis and the thickening is invariably uniform and smooth. In scurvy there may be a thickening of the long bones due to hemorrhage in the periosteum. In these cases the history and also the presence of other signs of scorbutus, such as hemorrhages in the skin or bleeding of the gums, will aid diagnosis.

Craniotabes.—In locating patches of so-called craniotabes, the surface of the occipital and other bones of the skull is examined for deficiency of bone formation. The occipital bone will in rachitis present membranous spots more frequently than is generally supposed. The most common tumors found on the scalp are those due to traumatism at birth, such as cephalohæmatoma, tumor of the scalp with depressed bone, and tumor due to syphilis. The cephalohæmatoma is found after birth and need not be described here. If an infant falls on one side of the head from a height, a depression of the skull may at once take place. This occurs if the bones are soft and not yet completely ossified. The depression is filled with an effusion of blood and serum. A soft tumor results which may not project above the surface at all or only slightly so. Around the border of the tumor the rim of bone bordering the depression can be felt. In this respect the condition differs from the cephalic hæmatoma found after birth. In the latter, the whole tumor is raised from the surface, and on physical examination there are no evidences of depression.

Syphilis (Fig. 83) may cause on the surface of the frontal and parietal bones tumors varying from the size of a hazelnut to that of a walnut. They may at first be hard and subsequently soften. They resemble abscesses, and should be differentiated from them. Tuberculosis of the bones may also cause such tumors. Tuberculosis of the skull bones in infancy is of rarer occurrence than syphilis of the skull, the cases of disease of the ear being excepted. In a concrete case, syphilis should be assumed until it can be excluded. Abscess may be diagnosed if there are abscesses elsewhere in the body. This is the case in folliculitis abscedens of Escherich. Mistakes rarely occur in these cases, since all the signs of abscess are present.

Acute Infectious Osteomyelitis.

Osteomyelitis is an acute infectious inflammation of the structure of the bones. It is common in infancy and childhood. Of 50 cases below the thirteenth year collected by Blumenfeld, 50 per cent. were under five years of age. The sexes were equally affected.

Etiology.—In the majority of cases the essential cause is the Staphylococcus pyogenes aureus. The disease may, however, be caused by any pyogenic micro-organism, such as the Streptococcus pyogenes, the pneumococcus, the Bacillus typhosus, the Recurrens spirillum, Bacterium coli, and the gonococcus. Of 90 cases collected and reported by Lannelongue, only 10 were due to the streptococcus. Lannelongue and Achard were the first to show that osteomyelitis may be caused by streptococci, in 1890. Van Arsdale and I, in 1891, published 4 cases of osteomyelitis caused by streptococci. These occurred in newborn infants or followed scarlet fever and pneumonia. The streptococcus osteomyelitis is of especial interest to the physician, as it occurs in infants and children under two years of age. It frequently follows infection of the umbilicus in the newborn infant, the exanthemata (scarlet fever and measles), and pneumonia. It differs from the staphylococcus variety in that the inflammation of the bone is less likely to involve the medullary canal, but affects the epiphysis. There is also involvement of the joints, with suppuration. The bacteria gain access to the circulation (Garré), and to the bones through some wound, such as the umbilicus; through the mucous membranes, as in ulcerations of the mouth; through some lesions of the skin, such as an eczema or furuncle, or through the gut. Of the 47 cases cited above, 17 were due to trauma, and 5 followed infectious diseases.

Pfisterer has recently published 7 cases of arthritis caused by the pneumococcus. In most of these cases the disease was monarticular; though in one case several joints were involved. The arthritis of this variety for the most part involves the larger joints. The symptoms are similar to the streptococcus form, and yield kindly to treatment. Some of the cases may complicate a pneumonia, or they may also occur independently of this disease. If complicating a pneumonia, the affection may appear from the first to the ninth week of convalesence.

Morbid Anatomy.—The seat of inflammation is the periosteum and the medullary substance of the bone chiefly. The inflammation of the marrow and spongy part of the bone involving the cortical bone layer is often spoken of as osteitis, that of the periosteum as periostitis. There is a primary form and one secondary to infections elsewhere in the body. It is a disease of young people and involves mostly the long bones. The periosteum is swollen, hyperæmic, the seat of hemorrhages and finally of purulent infiltration. The bone marrow and neighboring bone tissue is hyperæmic, the seat of hemorrhages, and after a time purulent infiltration. The whole marrow canal may be converted into a pus cavity, and pus may form underneath the periosteum. The bone tissue becomes infiltrated with pus, breaks down and forms sequestra. Abscesses may form in the bone. In the subsequent history of the separation of the diseased from the healthy bone the processes are those seen in all bone inflammations.

Symptoms.—In older children, the symptoms differ little from those of the adult subject. The femur and tibia are most commonly involved; next the humerus, superior maxilla, inferior maxilla, ileum, and radius, in the order named. In some cases the onset is sudden and the fatal issue takes place in a few days. In others, the invasion is gradual. In older children there are the regular symptoms of chill, fever, and vomiting, followed by local symptoms.

In young infants the signs of osteomyelitis are obscure. In the puerperal cases in newborn infants, the umbilicus may be inflamed for some days, after which the infant begins to cry when handled in the bath. One extremity is not moved and a joint may be swollen (Plate V.). Swelling of the joint may escape notice until the child is examined by the physician. After scarlet fever, the swelling of the joints is quite apparent, and also after pneumonia. In the newborn infant several joints may be swollen. In one of my cases in an infant ten months old, the elbow-joint and wrist-joint were involved, the whole radius being the seat of osteomyelitis. Similar cases have been published in this country by Gibney. The frequency of joint-involvement is a feature of osteomyelitis in children. Of 50 cases of osteomyelitis published by Blumenfeld, the joints were involved in 30. I have seen the multiple joint-suppurations most frequently in newborn infants. In all cases, there are evident swelling of the tissues about the joints and fluctuation in the joint-cavity. The joint contains pus.

The **diagnosis** is not difficult. If an infant cries when it is handled, every joint should be carefully examined. Osteomyelitis may be confounded with scorbutus. In the latter affection, the joints are painful and swollen, but do not contain fluid. In scorbutus there are ecchymoses, swelling and sponginess of the gums, and hemorrhagic lesions underneath the skin, all of which will aid in diagnosis. A history of umbilical inflammation or of scarlet fever is of great value. There are in congenital syphilis in young infants forms of inflammation about the joints which at first simulate osteomyelitis. In such cases the infant should be examined for other evidences of congenital syphilis, such as fissures and rhagades about the mouth and anus, mucous patches, and coppery discolorations of the skin. Tuberculous inflammation in the long bones or in the heads of the bones may present some difficulties of diagnosis. A study of the case and the absence of a history of acute trouble will solve the difficulty.

The **prognosis** of acute osteomyelitis in newly born infants is bad. The majority of cases are fatal owing to the formation of multiple foci of suppuration. The prognosis is also bad in infants under one year of age. The mortality of all cases under the fifth year is 56 per cent. In older children it is 20 per cent.

The **treatment** of acute infectious osteomyelitis is surgical.

SECTION XI.

DISEASES OF THE EAR.

Otitis in Infancy and Childhood.

Frequency.—Otitis media, catarrhal or purulent, is a very common disease of infancy and childhood. It is, as a rule, a secondary affection, but may in rare cases occur as a primary disease. Parrot first called attention to the frequency of otitis as a complication of bronchopneumonia. Netter made the first bacteriological examinations of the discharges from the ear. The subjects were 20 children whose ages ranged from nine days to two years. Kossel, Rasch, and Ponfick have investigated the frequency and nature of this affection in children. The results of their work show striking uniformity. Fully 85 per cent. of infants and children, examined post mortem, were found to have diseased ears. Most of the infants, especially in the material examined by Ponfick, had died of gastroenteritis, acute or chronic. Some had suffered from gastro-enteritis, pneumonia, or congenital syphilis.

The **etiology** of acute catarrhal, acute suppurative otitis media and of acute suppurative mastoiditis is much the same. The nasopharynx and the Eustachian tube are normally the habitat of various forms of bacteria. This is especially the case in infants and children who have enlarged tonsils and adenoid growths. A reduction of the vitality of the individual or any acute disease favors invasion of the ear by bacilli entering through the Eustachian tube. Thus the exanthemata, especially scarlet fever and measles, furnish a large quota of cases. Diphtheria, typhoid fever, typhus fever, varicella, influenza, gastro-enteritis, tonsillitis, and simple angina, also cause a large number of cases of otitis. Pertussis, cerebrospinal meningitis, and pneumonia are complicated by this disease. Sea-bathing, exposure to cold, and nasal douching favor its onset.

Bacteriology.—The bacteria found by different observers in the otitic discharges and in the cavities of the ear include the Staphylococcus pyogenes aureus, citreus, and albus, the Streptococcus pyogenes, the pneumococcus of Fränkel, the influenza bacillus and pseudo-influenza bacillus, the Bacillus fœtidus, and the Bacillus pyocyaneus (Netter, Kossel, Ponfick). The streptococci and influenza bacilli cause an especially severe inflammation, the pneumococcus a milder form. The diphtheria bacillus also causes otitis.

Morbid Anatomy.—In both forms of otitis and also in mastoid disease the tympanic membrane is injected and the vessels at its border are increased in size. The vessels of the hammer are injected. The epidermis of the tympanic membrane may be intact. The tympanic cavity may be filled with cellular elements. There may be a serous, mucous, purulent, or mucopurulent exudate. The mucous membrane of the tympanic cavity may be intact but injected, or may show gross defects. If the bony structures are involved, there will be necrosis of bone, especially of the tegmen tympani. There may be perforation of this structure or of the point of the mastoid process. The dura mater or sinuses of the dura may, in progressive mastoid, be inflamed. *There may* be cerebral abscess. If the pus does not escape by way of the Eustachian tube, it may perforate the tympanum. The exudate which fills the tympanic cavity contains epithelial cells, leucocytes, and blood-cells.

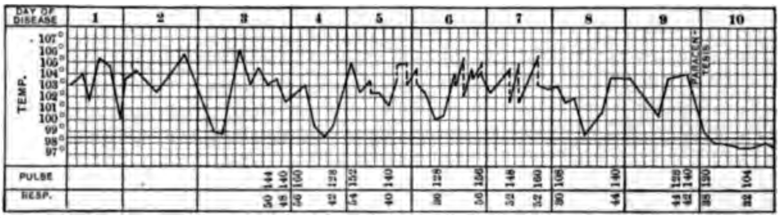
Otitis Media Catarrhalis.—Acute catarrhal otitis is, in a vast number of cases, simply a forerunner of otitis media purulenta or of an acute suppurative otitis. It will be convenient for the practitioner to consider these affections together.

They are more common among infants and children than among adults, and may occur at the earliest period of infancy. They occur most frequently in the spring and summer.

The causation has been considered under the etiology, and is the same in both affections.

Symptomatology.—In young infants and in children under two years of age, the symptoms are frequently masked by those of the primary disease. In many cases, the otitis gives no special warning of its presence. Perforation of the drum and a purulent discharge are the first intimation of the condition. This is especially the case in otitis in young nurslings who have suffered from acute tonsillitis or pneumonia, but these are not the cases which the practitioner is called upon to diagnose. In another set of cases, especially in those in which otitis is coincident with gastro-intestinal disorders of a chronic type, tending to atrophy, Heermann and Ponfick have shown that during life the otitis gives no objective symptoms although on otoscopic examination the tympanic cavity is found to be filled with pus. In cases which follow the milder types of influenza or angina, there may be a most puzzling set of symptoms which can only be referred to the ear. In these cases the physician finds, two or three days after the onset of tonsillitis or influenza, that the temperature does not drop to the normal; it may mount to 104° F. (40° C.) toward evening, and in the morning may drop to or within a degree of the normal. While the temperature is low the infant takes its food and plays. When it rises the infant becomes fretful, or stupid, or sleeps most of the time. There is no indication of pain. In some cases the infants perspire freely at the falling of the temperature. These simulate in many respects cases of malaria or of meningitis of the tuberculous type, except that the temperature rises higher than in the latter disease (Fig. 160). Local facial pareses may complete the resemblance to meningitis. The intermittent or recurrent curve of temperature may continue for a week or ten days. Only the careful exclusion of disease of other organs, and especially of the lungs and of the heart, will lead the physician to suspect disease of the ear. In nursing infants the bowels will be abnormal and the movements greenish, containing white curds. The temperature is, however, much higher than in any diarrhœa, and is more persistent and regular in its daily fluctuations. In cases of broncho-pneumonia complicated with otitis, previous to the spontaneous perforation of the drum the temperature will have shown more decided fluctuations than would occur at a late stage of the primary disease. However, in pneumonia there are few or no objective signs of the affection. Older children may have certain definite symptoms such as dull headache and pain in the ear, which, if sharp and stinging, will cause them to start in sleep, or to awake and cry out or put the hand to the ear. This last sign, so often mentioned in the text-books, I have seldom seen. There may be delirium and the fever may be quite high. Children, who can talk, complain of pain at night. There may be rushing, singing, or buzzing noises in the ear. Very characteristic is the starting of infants during sleep. Older children are out of sorts, and angry on awakening.

FIG. 160.



Otitis media purulenta in a child eighteen months of age. Symptoms and curve simulating closely a meningitis of the basal type.

Course.—Spontaneous perforation in a number of cases occurs in a few hours or a few days after the onset of the disease. As a rule, however, pain continues with fever until artificial paracentesis of the drum is practised. After spontaneous rupture of the tympanum, or paracentesis, the discharge may continue, being in some cases serous or serosanguinolent, and later becoming purulent. The purulent discharge may be profuse and the disease may advance into the mastoid or labyrinth. This frequently occurs in cases of the exanthemata or in pneumonia or influenza. In severe cases, the discharge may continue and become chronic, resulting in destruction of the structures of the ear. Complications may intervene, such as facial erysipelas, meningitis, cerebral abscess, thrombosis of the cerebral sinuses, and finally in suppurative cases pyæmia may intervene. On the other hand, after spontaneous rupture or paracentesis of one or both drums, the serous or purulent discharge may gradually cease and the ears be restored without any defect of hearing. In many cases incision of the drum in the very early stages of the disease is not followed by the discharge of pus; the symptoms cease, and the patient recovers. In other cases there is no rupture of the tympanum, although the tympanic cavity is filled with exudate, which discharges through the Eustachian tube. The pus may be swallowed and cause diarrhœa or pneumonia. In the cases of marasmus with otitis described by Heerman, the pus is believed to have found its way from the middle ear through the tube to the nasopharynx.

The **diagnosis** is first made from the rational symptoms. In my experience, the temperature-curve is a very useful guide in infants who give no evidence of pain. Otoscopic examination is the only positive means of making a diagnosis. There is congestion of the tympanum above Shrapnell's membrane and the long handle of the malleus. In the catarrhal cases the tympanum is red and angry or has a grayish lustre. The handle appears as a red or yellowish-white point. In some cases there are vesicles and interlamellar abscess. The exudate may cause bulging of Shrapnell's membrane or of the posterior-superior quadrant. Congestion remains long after resolution. In the suppurative cases the epithelium of the tympanic membrane may peel off. The tympanum is dull and lustreless. The auditory canal may be swollen. Perforation occurs, chiefly in the posterior-inferior quadrant. There may be pulsation of the membrane as well as bulging. The lymph-nodes beneath the ear may enlarge and that region may be very sensitive.

The **prognosis** in ordinary cases is good. In cases following the exanthemata it is grave, on account of the possibility of complications and of ultimate loss of hearing.

THE MASTOID REGION.

General Facts.—The mastoid region is important on account of the frequency of mastoid disease in infancy and childhood. In early life there is pneumatic tissue, but no mastoid cells are found. The mastoid process contains one large cell (Symington). The external wall is less thick and compact than in the adult. The petrosquamous suture is patent. The petrosquamous sinus is persistent in some cases, passes through a foramen on the inside of the skull, and appears externally behind the glenoid fossa and tympanic ring. Thus infectious material may easily be conveyed internally. In infants and children pus finds its way externally more readily through the open fissura mastoideo-squamosa.

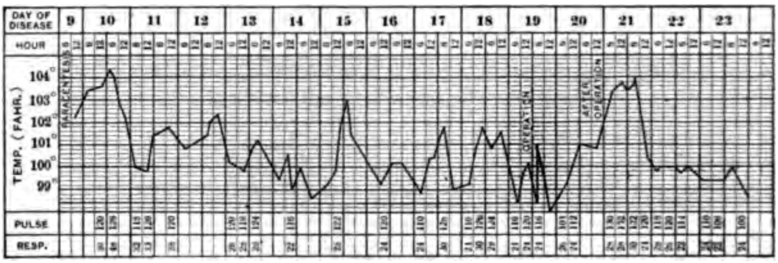
Etiology.—Inflammation of the mastoid is rarely primary. The mastoid may at the outset be inflamed when there has been no antecedent otitis. As a rule, however, inflammation of the mastoid is secondary to acute or chronic otitis. The causation is identical with that of acute or chronic otitis.

Of 39 cases of mastoid disease under eight years of age, collected by Knapp, 7 occurred in the first year, and 9 in the second. The greatest frequency is therefore after the second year. It may occur as early as the second month. I have had a case in an infant three months of age. The anatomical conditions favor the occurrence of mastoid disease in infancy and childhood. The Eustachian tube is short and of large calibre; infectious material from the nasopharynx can easily gain access to the ear.

Symptoms.—Clinically, mastoid disease in infancy and childhood manifests itself by rational symptoms and physical signs. There may be extensive mastoid disease without any external physical signs. In one of my cases of otitis, which was observed from the outset, extensive mastoid disease in a child of three years of age did not give any external signs. The clinical symptoms are characteristic. The drum may have been perforated after otitis, or paracentesis may have been performed. After perforation, the temperature present during the preceding otitis drops to the normal. The patient is able to be up and about. The ear discharges freely. After two or three weeks there is a sudden or gradual rise of temperature, which may be slight or may reach 103° or 105° F. (39.4° to 40.5° C.). There is restlessness at night. On inspection, the ear may not show anything abnormal. The temperature, however, continues to be remittent for several days. On otoscopic examination, there is found to be swelling of the roof of the auditory canal or of the floor of the attic. In other cases, after a very early and timely paracentesis of the drum, the patient does not do well. The child is restless at night, at intervals irritable and then playful, and starts from sleep (Fig. 161). The temperature fluctuates daily from 100.8° to 102° F. (38.8° C.). On some days it may be normal or subnormal. The ear discharges for days, but a slight temperature continues. If the patient is an infant or a young child, it may be very difficult to ascertain whether pain is present on pressure backward over the region of the antrum behind the ear. There is in early cases no swelling or redness behind and above the auricle. As was stated above, there may be extensive and advanced mastoid disease without external redness or swelling. In such cases the lymph-nodes behind the ear and at the angle of the jaw may be swollen and painful. Young children and infants do not complain of pain. It is only in older children that it can be noted.

Mastoid disease which follows the exanthemata, especially scarlet fever or measles, or occurs late in typhoid, shows certain characteristic clinical features. During the fifth or sixth week of scarlet fever the ears may discharge profusely. There is a daily rise of temperature in the afternoon, which is slight in some cases. The patients play in the early portion of the day, but in the afternoon appear listless, and have a slight frontal headache. As days pass, the children become stupid during the afternoon rise.

FIG. 161.



Otitis media in a female child, three years of age. Observed from the onset. Early paracentesis, fall of temperature, then rise again. Subsequent mastoid involvement necessitating operation.

In many cases of scarlet fever, as may be seen by referring to Figs. 30 and 31, otitis is a complication. The temperature does not fall to the normal, as it should, after the fading of the eruption. There is slight aural pain at night, which is sometimes sufficiently severe to deprive the patient of sleep. In other cases the temperature drops to the normal and suddenly rises in the second week. In both these sets of cases there is an otitis which may develop into mastoid disease, or in which mastoid disease may have been present from the outset.

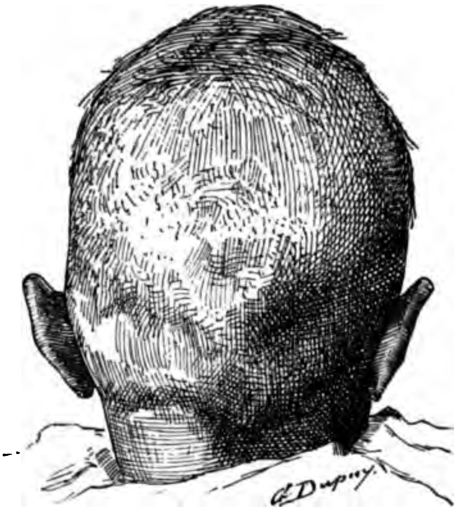
Körner calls attention to the fact that in late typhoid fever, chills, with rises of temperature, may be, in the absence of other signs, indicative of serious mastoid disease.

Physical Signs.—**Pain** is a physical sign of mastoid disease in children. In most cases it cannot be elicited by the most skilful manipulation. In others it is impossible to come to a definite conclusion. In older children pain may be elicited by pressing the mastoid bone in a backward direction, care being taken not to press on the auricle. The pressure should be firm and continuous. Pain in the tip of the mastoid is not of value unless there has been a perforation and phlegmon at that point (Dench).

Otoscopic Examination.—There is a shortening of the external canal in its posterior and upper aspect (Dench). The upper posterior wall sinks. There is bulging of the upper portion of the tympanum.

Tumefaction posteriorly and above the ear occurs in infants only in neglected cases. According to Dench, in these cases the pus escapes from the antrum through the aditus ad antrum into the tympanic vault. It then finds its way through the Rivinian fissure along the upper wall of the canal to the external surface of the mastoid. In children cases in which this swelling appears are less serious than adult cases. The swelling also appears much earlier in infants and children.

Fig. 162.



Mastoid disease in a child eighteen months of age. Swelling behind the ear over the mastoid. The ear is displaced away from the scalp.

Diagnosis.—The life of the patient often depends upon the early recognition of mastoid disease. The diagnosis in infancy and childhood should not only be made early, but should be made chiefly from the clinical symptoms of temperature, which will in its fluctuations show a septic curve. The history of the case is of service. Presence of pain is of no value in infants and young children. The daily otoscopic examination of the discharging ear will give positive evidence of mastoid disease. The signs detailed in the paragraph on symptoms are of great importance. A profuse discharge does not preclude mastoid disease. Facial paralysis is of no value. I have seen it in cases in which mastoid disease was on operation found to be absent. Tumefaction is seen only in late cases. Redness is sometimes apparent before the appearance of swelling behind the ear.

Course.—If a case is neglected, pus from the mastoid may force its way through the tympanic roof and cause cerebral abscess or meningitis. It may destroy the plate (lamina vitrea) of the sigmoid sinus and cause thrombosis, may find its way through the tip of the mastoid along the border of the sternomastoid, and cause phlegmon, or may force itself through the sutura mastoideo-squamosa, causing swelling behind the auricle.

Treatment.—**Prophylaxis.**—Children can be taught to tolerate the therapeutic measures which, if catarrhal inflammation of the fauces is present, as in the exanthemata, will cleanse the parts. Thus in scarlet fever, an intelligent child will readily allow the throat to be sprayed with normal saline solution. Swabbing the throat or applying any drug locally is impracticable in children.

If the pain is excessive a mild opiate, such as paregoric, is administered. In young infants the severity of pain cannot be estimated. In older children dry heat applied externally to the ear by means of a water cushion relieves the pain. Some authors advise the application of leeches behind the ear, or the instillation of water at 110° F. (43.3° C.) into the canal with a dropper. Inflation of the ear in the early stages of otitis media has been advocated and condemned. Suction by means of a catheter introduced into the Eustachian tube is also practised. If the pain and fever are not relieved by these measures, incision of the drum is resorted to. Whether the otitis is catarrhal or purulent, paracentesis is best performed early, since damage to the ear may thus be avoided. The method of performing paracentesis of the drum is best learnt from special text-books on the subject. Duel advises enlargement of the opening in cases in which spontaneous rupture of the drum has taken place. Drainage by the introduction of sterilized absorbent gauze into the canal is superior to syringing. If this is not possible, syringing with 1 : 5000 bichloride is useful.

The indications for the performance of mastoid operation are protracted otitis with profuse otorrhœa, there being no tendency to resolution, acute otitis in which there is a tendency to resorption and in which paracentesis has not established drainage, also mucopurulent otitis maintained by mastoid involvement, otitis with symptoms pointing to meningeal complications, and finally otitis with complicating stenosis of the external canal, preventing drainage.

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SECTION XII.

DISEASES OF THE KIDNEYS AND UROGENITAL TRACT.

THE weight of the kidneys is $\frac{1}{120}$ of the body weight in the infant and $\frac{1}{240}$ in the adult.

It is not, as a rule, possible to palpate the normal kidney in the infant or child. I have, however, seen in young infants exceptional cases in which the kidneys were situated very low down and could be easily palpated through the abdomen. I have found floating kidneys in infants and older children, but not so frequently as other observers. Comby in 1898 reported 18 cases, of ages ranging from one month to ten years of age. Steiner, Stewart, and Abt have also reported a number of cases. I believe that the displaced and fixed kidney is congenital. As the child grows and the parts are stretched, the attachments of the kidneys, congenitally low, become more relaxed. This would account for a number of cases. Jacobi believes that floating kidney in children is a congenital anomaly.

Sixteen of Comby's cases occurred in girls. A displaced, fixed kidney in infants causes no symptoms. In cases of movable kidney or floating kidney the main symptom is pain, either epigastric or radiating from the iliac region. In a girl of eight years with floating kidney, there was no difficulty in palpating the enlarged movable kidney below the liver. There were attacks of acute colicky epigastric pain, which occurred independently of the ingestion of food. The child was nervous and hysterical.

CYCLIC ALBUMINURIA.

(*Postural Albuminuria.*)

Cases of this form of albuminuria were first published by Vogel, Ultzmann, Gull, and Leube. The systematic description was first given by Pavy, by whom it has been carefully studied.

Cyclic albuminuria occurs principally in children and adolescents; 40 per cent. of the cases occur in children from the first to the fifteenth year, and 80 per cent. of the cases occur before the twentieth year. It is, therefore, distinctly a disease or condition observed in a period of metabolic activity and growth.

The characteristic symptom is the appearance of albumin in the urine in the forenoon and afternoon, and its disappearance after a night's rest in the recumbent position. It is not present in the morning directly after rising, but appears soon after the upright position has been assumed. The quantity of albumin is not excessive. It may disappear from the urine for days and weeks, and again reappear. The quantity of albumin does not progressively increase. The urine never contains formed elements of the kidney, such as casts.

There is no doubt as to the existence of this form of albuminuria in children, but its significance is a matter of wide diversity of opinion. Heubner has lately published some cases, and has collected from the literature 22 cases in children from one to fifteen years of age. Some authors, among them Heubner, are inclined to give a good prognosis in these cases, and to regard them as physiological forms of albuminuria. Others, among them Henoch, Leube, and Senator, are inclined to regard them as due to insidious changes in the kidney following infectious disease. It should be remembered that after influenza, scarlet fever, or diphtheria, small quantities of albumin are, at intervals, present in the urine for months and years. There may also be occasional hyaline or epithelial casts and a few blood-cells. These disappear either with or without treatment of diet and rest, but later reappear. I have seen this occur in children in good health. More careful data on the subject are needed. In a given case the urine should be carefully and frequently examined for kidney elements. It should be remembered that, in nephritis, the albumin in the urine frequently takes a cyclic course (Senator).

So long as there is albumin in the urine, the prognosis must remain conditional on the prolonged observation of the patient, for a few of the published cases have in later years developed nephritis. It is said, also, that this form of albuminuria has been met in several members of the same family, and in families in which albuminuria and nephritis have existed. The term cyclic albuminuria should be limited to those cases in which there has never at any time previous to or during observation been any form-elements of the kidney in the urine. All the cases published, and those which I have observed, occurred in children with lymphatic constitutions. They showed a marked anæmia at times. There was an œdema of the face but not of the extremities. The children complained at various times of headaches or a heavy feeling in the occiput, were easily tired, awoke feeling tired. They were subject to dreams and were of a nervous temperament. In one of my supposed cases the child was free from the above symptoms, and was the picture of health. In this case there were periods early in the disease in which very scanty form-elements occurred in the urine with the albumin; at others, none could be found. The case was at first diagnosed as cyclic albuminuria; but my fears have been justified, inasmuch as lately the form-elements, such as casts and blood, have increased in the urine and have become permanent, thus showing the danger of diagnosing these cases on short periods of observation. Two cases which I have now under observation are classic, in that many repeated examinations (extending over a year) of the urine have failed to reveal anything pointing toward an affection of the kidney. The albumin is present some time after rising in the morning, and after exercise. It disappears on enforced rest.

Treatment.—It has been proposed in cases of cyclic albuminuria to enforce at intervals periods of rest of one or two weeks at a time, and the limitation of exercise and sports. I have tried this method, at the same time dieting the patient, but have not found it as successful in improving the general condition of the patient as moderate outdoor exercise in the open high country—freedom from mental worry, such as the suspension of studies; good, simple food; perhaps a tonic of the iron series. City and school life are not conducive to aiding the physician in treating these cases. A persistent anæmia sets in under these conditions, and is the symptom that baffles the physician in the treatment. Nothing will improve the patient so much as outdoor exposure in the open country.

ŒDEMA OR HYDRÆMIA WITHOUT KIDNEY LESION.

Weak infants who have suffered from chronic gastro-enteric catarrh have swelling or an œdematous condition of the dorsum of the feet and ankles. There may be slight anasarca elsewhere. There is no real kidney lesion; the condition is one of hydræmia. The changed state of the tissues, including the vessels and blood, allows of a transudation of serum into the subcutaneous structures. On examination, the urine is found to be abundant and of low specific gravity, but without evidences of nephritic degeneration. In children of two years of age this condition of slight subcutaneous œdema occurs in simple anæmia of a severe type. In these cases the skin is yellowish, the ears have a waxy clearness, the eyes have an œdematous appearance, and the lips, hands, and feet are puffy. The condition is known as hydræmia or hydræmic anæmia.

DYSURIA.

Dysuria, or difficult and painful micturition, is a condition in which there is partial obstruction to the free flow of urine from the urethra. It is not uncommon in young infants and children, and may be due to a variety of causes. If lithiasis is the cause, there is not only pain in passing the urine, but there may, in the intervals, be acute attacks of pain, due to the passage of calculi along the ureter. Examination of the urethra in the male often results in finding a calculus of very small size in the anterior penile urethra. In lithiasis, there is sometimes very painful micturition without the formation of calculi of any size. The minute crystals of uric acid cause a smarting sensation as the urine passes over the urethra. In febrile states with concentrated urine, the acidity of the urine, and the excess of uric acid with free crystals, cause painful micturition.

Simple or gonorrhœal inflammation of the urethra may cause difficult and painful micturition. Dysuria is painful at the onset of vulvovaginitis. Another condition of congenital origin, which was described by Bokai as **cellular atresia of the labia,** is a very common cause of dysuria. It is seen in very young female infants. From birth, the urine is passed in drops and with great straining and pain. In some cases it is passed without pain, but the condition of atresia attracts attention. On gently separating the labia majora a thin pinkish-white membrane is seen to occlude the introitus vaginæ completely. At the urethral end of this membrane, a very minute opening is seen, through which the urine filters. These membranes can be divided by means of a dull director. It is then seen that the hymen and urethra are directly behind the membrane. The operation of dividing the membrane is exceedingly simple, and causes little or no bleeding. Bokai has described a similar condition in boys, which is somewhat less common. It is a cellular adhesion of the prepuce and glans penis which not only causes false phimosis, but also difficult and painful urination. He found that in the newly born infant the prepuce was sometimes adherent to the tip of the glans penis, and that across the opening of the meatus there was a very thin membrane. In other cases, this membrane was ruptured, but the prepuce still remained adherent to the glans in front, while behind at the corona glandis there was retention of smegma and consequent painful inflammation.

The **treatment** is division and separation of the cellular adhesions. Other abnormalities in infant boys, among them diverticula of the urethra, may cause dysuria.

HÆMUTURIA.

Hæmaturia is the passage of blood and its elements into the urine, in which blood-cells and coloring-matter are found. The condition may occur in the following states:

(*a*) Acute nephritis of all forms, especially those complicating the infectious diseases, such as scarlet fever, measles, typhoid fever, and malarial fever.

(*b*) Calculi, renal or vesical.

(*c*) Malignant growths of the kidney—sarcoma and carcinoma.

(*d*) Growths of the bladder—polypus.

(*e*) Traumatism in the region of the kidney.

(*f*) The ingestion of drugs.

(*g*) Scorbutus.

The color of the urine varies from a slightly smoky amber to a deep brownish-red. There may be a deposit of blood-cells and clots in the urine. Pure blood with clots is seen in cases of malignant tumor of the kidney and calculi of the kidney or bladder. Smoky urine is seen in cases of nephritis and drug-poisoning.

HÆMOGLOBINURIA.

Hæmoglobinuria is a condition in which the urine contains the coloring-matter of the blood, but, except in rare cases, no red blood-cells. The urine is reddish or brownish, and has a high specific gravity. It contains albumin. By spectral analysis, the spectrum of the blood coloring-matter is obtained. According to Hoppe-Seyler, methæmoglobin and not hæmoglobin is often the coloring-matter present. There are few blood-cells and no detritus. Several theories have been advanced to explain the appearance of hæmoglobin in the urine, that of Ponfick being generally accepted. According to that author, either the blood-cells are destroyed by some vicious agent or ferment (Ehrlich) and the hæmoglobin is thus let loose into the circulation, or the hæmoglobin is dissolved out of the blood-cells and passes into the circulating plasma, leaving the cells behind as so-called "shadows." Whatever the real cause, the exciting influences are as follows:

(*a*) Cold or exposure to wet. Hoff and Demme have published cases of children with paroxysmal hæmoglobinuria following such exposure.

(*b*) Drugs, such as arsenic, phosphorus, potassium chlorate.

(*c*) The infectious diseases, such as malaria and scarlet fever, erysipelas.

(*d*) Hæmoglobinuria has been observed in cases of burns.

(*e*) Baginsky has observed hæmoglobinuria in children with nematodes.

One-half the cases published have a history of syphilis. Such is the case published by Hermann, occurring in a boy four years of age, with a history and physical marks of congenital syphilis. In this case the boy had at times attacks of hæmoglobinuria.

In the paroxysmal form, each attack is preceded by a chill and followed by dyspnœa, palpitations, cyanosis, and severe symptoms of collapse. The attack may last a few hours or a few days, the duration depending on the course of the primary affection. This form has been especially observed to occur in pernicious malarial fever.

The **prognosis** is very good. Patients quickly recover from the attack proper, and there is no danger to life. The cases of syphilitic origin are not controlled by antispecific treatment, though the condition of the blood is improved. Chvostek succeeded in abating an attack by the administration of amyl nitrite.

Morbid Anatomy.—Dieulafoy and Widal found in a fatal case the cortex of the kidney dark brown in color; the cells of the glomeruli were normal. The cells of the convoluted tubes and the tubes of Henley were infiltrated with pigment-granules, which were also present in the lumen of the tubes.

The **treatment** consists not only in the management of the primary exciting conditions, but, if there is a history of syphilis, an antispecific course of treatment is indicated. With this we may give tonics, such as iron, and exert a certain amount of prophylaxis by protecting the patient from cold, and also, if possible, securing to the patient wholesome food.

RENAL CALCULI.

(*Uric Acid Infarction; Lithæmia.*)

So-called uric acid infarction is found in the kidneys of over one-half the infants who die in the first weeks after birth. These infarctions are seen in the medullary portion of the kidney as golden-yellow or brownish rays which are broader toward the papilla. Ebstein found isolated deposits in the cortex. The infarctions consist of uric acid (Schlossberger). They are supposed to be due to the destruction of tissue rich in nuclein (cells) (Kossel and Horbaczewski). They are found in weaklings, and more often in infants who have been born living than in stillborn infants. During the first weeks of life they are washed out by the urinary secretion. Hence the increased uric acid excretion at that time. As a rule the condition gives no symptoms. It is not uncommon for the diapers of the infants to be stained red, and in older children there may be the so-called brick-dust deposit in the urine. In these cases there may be a history of severe colicky attacks. In other cases the infant or child experiences pain on urination and cries piteously. Some older children will run about in pain and grasp the penis. In all such cases I examine the diapers for concretions. Failing to find these, I carefully examine the urethra. In several cases I have found an oval calculus of the size of a rice-seed, imbedded in the canal of the penile portion of the urethra. These cases have attacks of pain extending over months, and probably caused by the passage of the calculi from the kidney through the ureter, the bladder, and urethra. The calculi are easily extracted with long-bladed forceps. In one of my cases of hæmaturia, in a boy three years of age, there were several attacks lasting for days, but no distinct history of pain. The urine contained blood coloring-matter, some blood-cells, and a few hyaline casts, which it was difficult to find. The diagnosis was obscure until a few small calculi were found in the urine. Urotropin given in small doses caused a cessation of symptoms.

ACUTE NEPHRITIS.

A. Acute Parenchymatous Nephritis or Acute Exudative Nephritis (DELAFIELD); Tubular or Glomerular Nephritis.

B. Acute Diffuse Nephritis or the Acute Productive Nephritis (DELAFIELD).

The **etiology** of both forms of acute nephritis is the same.

There is scarcely an acute infectious febrile disease which may not give rise to acute nephritis. It complicates or follows scarlet fever, measles, influenza, diphtheria, infectious angina, pneumonia, rheumatism, typhoid fever, sepsis of all kinds, variola, parotitis, malaria, and congenital syphilis. The frequency in scarlet fever of the œdematous forms with anasarca has led to the belief that this disease was most often complicated by nephritis. If the parenchymatous form is included, the condition will be found to be very frequent in other infectious diseases, but it is often unrecognized. The essential causes of acute nephritis are micro-organisms or their toxins. Thus in the various diseases, the Diplococcus pneumoniæ, the typhoid bacillus, streptococci of various kinds, staphylococci, and the Bacillus pyocyaneus, have among other bacteria been found in the kidney. On the other hand, in diseases such as diphtheria, the toxins of the bacteria are the cause of the parenchymatous or diffuse nephritis (Fürbringer, Roux, Councilman). If the toxins are formed in the body, the infections are said to be autochton or endogenous. The irritating toxin may be introduced from without, as in chloroform or ether narcosis, and the ingestion of drugs (ectogenous). The rôle played by cold as a causative factor is still a matter of speculation. Its mode of action, whether reflex, through the circulation, or by causing changes in the blood, is still obscure.

Morbid Anatomy.—**Acute Parenchymatous or Exudative Nephritis** (Delafield).—This is an acute inflammation of the kidney, in which the principal changes occur in the epithelium of the tubules and Malpighian tufts. The kidneys are larger than is normal, and succulent. The capsule can be stripped from the surface, which is red, grayish, and punctate in spots. All the changes are most marked in the cortex of the kidney. Evidences of inflammation are found in the tubes, stroma, and glomeruli. The epithelium of the tubes is flattened, granular, and fatty, or in a condition of coagulation-necrosis. The lumen of the tubules may be empty or may be filled with desquamated epithelium or with coagulated masses (casts) of a hyaline character. Delafield describes the tubes, in severe cases, as filled with leucocytes and blood-cells. The tubes may be uniformly dilated.

The changes in the glomeruli may be so slight as to be scarcely noticeable. The cavities of the capsules sometimes contain coagulated matter and red and white blood-cells (Delafield). In marked cases, there are desquamation of capsular epithelium and increase of nuclei. The swelling and proliferation of cells sometimes change the appearance of the tuft so that the outlines of the individual capillaries are lost. The stroma is infiltrated with serum, and in severe cases there are in the cortex small collections of white blood-cells (pus).

The changes in **acute diffuse nephritis**, or the acute productive nephritis of Delafield, are more serious and permanent. According to Delafield, the kidneys are large, and at first smooth and later rough. The cortex may be mottled yellow and red; the pyramids are red. In this form of nephritis there are the changes found in exudative nephritis, and also a growth of connective tissue in the stroma and an increase of the capsule cells of the Malpighian bodies. These changes involve symmetrical strips in the cortex, which follow the lines of the arteries (Delafield). The Malpighian bodies show an enormous growth of capsule cells with compression of the tufts. If the nephritis is acute, the interstitial tissue is augmented with newly formed cells and basement substance. There is a new growth of connective tissue between the tubules; the walls of the arteries are thickened. In the capsule of the Malpighian tuft, there is a growth of cells which compress the tuft of vessels. These and the vessels are in turn converted into small balls of fibrous tissue (Delafield). In addition there may, in the acute forms of nephritis, be hemorrhages throughout the kidney substance.

Symptoms.—In the forms of parenchymatous nephritis which complicate the febrile infectious diseases, influenza, pertussis, angina, and gastro-enteritis, either the symptoms of the primary disease mask those due to the kidney lesion or the nephritis may be so mild as to give no symptoms. Thus in the parenchymatous nephritis which complicates or follows influenza, there are after the attack has passed no symptoms referable to the kidneys, yet on examination the urine shows a trace of albumin, hyaline and a few epithelial casts, and an occasional red blood-cell. In these cases there is no œdema of the tissues, no headache, and the children are apparently well except for the changes in the urine. These may at first be quite marked. After a few months the albumin may only appear occasionally; the casts and blood disappear for weeks and then reappear. For weeks or months the children may have no constitutional symptoms. In the parenchymatous nephritis, which is seen in severe forms of gastro-enteritis and dysentery, the signs in the urine of marked nephritis are albumin, casts of all kinds, and blood-cells (Parrot, Fischl, Czerny, Koplik, and Morse). Although Czerny traces a certain form of dyspnœa to the influence of uræmia in these cases, no distinct set of symptoms due to the kidney can yet be formulated. It is true that there are terminal anasarca, suppression of urine, and vomiting, but the presence of all these may be explained by the severity of the intestinal lesions.

Changes in the Urine.—In all the diseases above mentioned, the parenchymatous nephritis may in infants and children be evinced by diminution of the quantity of urine, or the presence of a trace of albumin, or a few hyaline or epithelial casts and blood-cells. The quantity of urine may, however, be normal. In other cases, the albumin is more marked and the casts much more numerous. Renal epithelium is also present. Leucocytes are rare.

In the diffuse or productive form of nephritis in infants and children, the symptoms are marked. In some forms of nephritis complicating scarlet fever the lesion never advances beyond the parenchymatous stage, and at that period the symptoms are either not present or not noticeable. If the nephritis is more marked, however, it is noticed at the end of the third week that the patient is somewhat pale, that the face is a little swollen especially about the eyes, and that there is very slight œdema of the general surface. In these cases it is possible at the end of the period of eruption, to find a slight trace of albumin in the urine and a few hyaline and epithelial casts. With the onset of the anasarca the albumin increases in quantity, the casts in number, and a few blood-cells are found. The quantity of urine is diminished, but in the mild forms not markedly so. A boy of six years may pass half the normal quantity. There is no headache, and only a few obscure pains in the joints. There is occasionally slight pain in the region of the kidney. The temperature is normal or may at intervals of several days rise a degree or a degree and half above the normal. The nephritis is probably of the mild diffuse type. In three weeks the mild anasarca disappears, the anæmia improves, and the urine becomes normal.

In the more severe cases there is a rise of one or two degrees in temperature, and the patients have marked general anasarca. If old enough, they complain of headache, they vomit, and show marked decrease in the number of respirations and pulse, the irregularity of pulse being of a purely uræmic character. In some cases there are effusion into the chest (hydrothorax) and abdominal ascites. The quantity of urine is much diminished, there being only one or two ounces in twenty-four hours. The specific gravity is high; the urine contains blood, leucocytes, and casts (hyaline, granular, and epithelial), with blood cells. Under treatment, the vomiting, headache, and anasarca subside, the quantity of urine increases, the number of casts and blood-cells diminishes, and the patient makes a good recovery. In other cases the initial anasarca becomes more marked, there being considerable œdema of the whole surface; the urine is entirely suppressed; the vomiting and headache increase; convulsions set in; there are several attacks of eclampsia; the patient becomes comatose, and may die of uræmia, or after one or two attacks of eclampsia, the symptoms may abate and recovery take place.

There is a very fatal form of diffuse nephritis which occurs on the fourth or fifth day of malignant scarlet fever. On the third day, at the height of the eruption, the patient passes into a delirious, semi-conscious state. The quantity of urine is much diminished; its specific gravity is high; casts of all kinds and blood are present. The urine may finally be totally suppressed. There is no œdema of the surface. Coma and convulsions set in. The patient succumbs to the intense general toxæmia and to its effect on the kidneys. In these cases the kidney symptoms cannot be separated from those caused by the general intoxication.

Individual Symptoms.—THE VOMITING.—The vomiting in scarlatinal nephritis is rarely distressing, and subsides in a short time. It is not a constant symptom, nor is it of serious import.

The HEADACHE is not a very marked symptom in children.

ŒDEMA is present in a large proportion of cases, and is marked in the severe ones. It may occur with hydrothorax, ascites, and hydropericardium. It may affect only the face, or the lower extremities alone. It may be so intense as to cause bursting of the skin and the escape of serum through the fissures. It may affect one half the body more than the other (Henoch). Under all these conditions, the outlook is serious.

The PULSE is sometimes inordinately slow. It may be more rapid than normal, and may show marked irregularity.

The HEART may, as was pointed out by Henoch and Friedländer, be the seat of hypertrophy and dilatation. There may be complicating endopericarditis.

The LUNGS may be the seat of pneumonia, or œdema of the lungs may suddenly develop. There may be complicating pleuritis.

There may be CONSTIPATION or more or less diarrhœa.

There are cases in which the TEMPERATURE is normal or subnormal during the whole course of the disease. In the cases in which there are sudden eclamptic seizures, the temperature may mount to 104° F. (40° C.) during the attacks. On account of the rupture of a bloodvessel in the brain during the eclamptic seizures, there is in many cases, after the subsidence of the uræmic symptoms, aphasia, or hemiplegia of a more or less permanent nature.

Patients with nephritis succeeding scarlet fever develop fainting spells with cyanosis, galloprhythm, and all degrees of cardiac weakness. It is difficult in such cases to know whether to attribute these symptoms to the nephritis or to myocarditis which is the result of the scarlet fever.

THE URINE.—The general characteristic features of the urine in acute diffuse nephritis of scarlet fever have been given. Suppression may take place suddenly. The urine may not have contained coagulabie albumin or casts, and the quantity may have been normal. The common notion that uræmia or eclampsia can supervene only if the quantity of urine is diminished, is erroneous. Even if the quantity is above the normal and the urine contains little albumin and few casts, eclampsia may supervene with fatal results. An increase in the quantity of urine above that of the normal is an unfavorable symptom. The quantity of urea passed is always the crucial test. There are cases in which blood appears in the urine and in which there is true hæmoglobinuria, which may give rise to irritation of the kidney. In other words, the hæmoglobinura is primary, the nephritis secondary. The quantity of albumin in the urine varies greatly; it may only amount to a trace or be sufficient to cause the urine to become solid when boiled.

The Primary Forms of Acute Nephritis.

The question has arisen: Can nephritis be primary? If nephritis is the result of some form of infection, it cannot be primary. Henoch, Heubner, Bouchut, Bartels, Loos, and Holt have published cases in nurslings, the origin of which could not be traced. These occurred in infants from five weeks to one and a half years of age, who suddenly developed marked anasarca and vomiting, with suppression of urine. Some of the cases had a febrile movement of a remittent type. The majority of them were fatal. Their exact nature is still unknown. Uhlenbrock has recently collated all the cases in the literature, but has thrown no light on the subject. On autopsy, a few cases have shown a parenchymatous nephritis.

Course.—The majority of cases of parenchymatous or exudative nephritis recover. The prognosis of the diffuse or productive form is more serious, but in exceptionally mild cases recovery may take place. Other cases make an apparent recovery. After the symptoms of œdema and anasarca have disappeared, anæmia remains. The albumin in the urine may disappear and reappear. In six months or a year, general anasarca may set in with all the symptoms of an acute exacerbation of the disease. The patient may eventually recover from the attack, but as a rule others of the same kind follow, and the condition of chronic nephritis results.

Duration.—The acute forms of parenchymatous or diffuse nephritis last from two to six weeks. The parenchymatous forms are sometimes evanescent, the marked symptoms lasting only a week.

Chronic Diffuse Nephritis.

(*a*) *Chronic Productive Nephritis*, (*b*) *Chronic Nephritis without Exudation* (DELAFIELD).

The forms of chronic diffuse nephritis are the same in childhood as in adult life. They usually occur late in childhood. Thus one case of chronic diffuse nephritis in a girl of fourteen years of age dated from an attack of scarlet fever at the age of eight years. At autopsy there was found a diffuse nephritis of the productive variety (large white kidney). In another case, a boy of twelve years, with diffuse nephritis of the non-productive variety (small cirrhotic kidney), had had an attack of scarlet fever at the age of five years. He had no anasarca in the course of the nephritis. Active symptoms of headache and vomiting appeared a year and a half before his death. The quantity of urine was above the normal and there were a few hyaline casts. At autopsy a small kidney was found. Thus there may in children be two forms of chronic nephritis following scarlet fever or any other infectious disease. Adults present symptoms referable to the eye, such as neuroretinitis, which I have not met with in children, and which must be exceedingly rare. Neither have I seen in children the emphysema met in adults. The heart may be hypertrophied and dilated in children as in the adult. They may have endocarditis and pericarditis with pleurisy.

Treatment.—The forms of parenchymatous or exudative nephritis which so frequently occur as accompaniments of the acute febrile disorders, pneumonia, typhoid fever, influenza, etc., need little or no treatment. There are no symptoms referable to the kidney. Nephritis accompanying acute gastro-enteritis is best treated by remedies directed toward the primary affection. The quantity of urine is sometimes diminished. It contains casts of all kinds. Rectal enemata of saline solution at a temperature of 108° F. (42.2° C.) are then of great utility, not only in supplying fluid to a depleted circulation, but also in stimulating the circulation and therefore the kidney secretion. Drugs which might still further compromise the condition of the kidney should not be given for the intestinal affection. Hot baths are of great utility, 105° F. (40.5° C.).

In the partial or complete suppression of urine seen in the first few days of the malignant forms of scarlet fever, more active treatment is required. When the temperature is high, the pulse rapid and weak, the patient unconscious or delirious, and the urine diminished or suppressed, I administer high and large rectal enemata of water at a temperature of 108° to 110° F. (42.2° to 43.3° C.), as recommended by Kemp. These should not be given to children with a double-current tube, but simply as enemata. About a quart of saline solution is thrown into the rectum at very low pressure. A fountain bag syringe is utilized for this purpose. These enemata stimulate the heart and circulation and supply the system with normal fluid. To stimulate the skin, the warm baths are preferable to cold ones. Patients are frequently much depressed by cold packs or baths given to reduce the temperature. The temperature of the bath should be at least 105° F. (40.5° C.), and the patient allowed to remain in it five or ten minutes, according to the state of the pulse.

In acute cases the anasarca will, as a rule, take care of itself. If it is extreme, Senator advises the administration of diuretics in acute as well as chronic nephritis. Some authors recommend diuretin and digitalis in form of infusion, a drachm being combined with an agreeable alkali, such as citrate of potassium. The pulse should be watched. If it is low, the digitalis is suspended. I do not utilize whiskey or alcohol in these cases. In acute diffuse nephritis and in productive nephritis similar to that of scarlet fever, the uræmic symptoms, the œdema, and the kidneys are treated. Vomiting is a uræmic symptom which is prominent at first. If the patient vomits everything ingested, no food should be given by mouth. The patient is nourished by rectum by means of somatose or nutritive enemata. The headache needs little treatment. Bromide and a small dose of chloral or trional are given for restlessness at night. In the forms of nephritis, generally subacute, in which there are œdema amounting to anasarca, and diminution of urine, baths and diuretics are beneficial. The anasarca is sometimes scarcely noticeable, and the quantity of urine little diminished. There are usually a few hyaline and epithelial casts, and also blood-casts. The patient is kept in bed and put on a milk diet. The bowels are kept open by means of Vichy water given in liberal quantities, or by Carlsbad salts. A child between four and six years of age should take half a drachm of the salts once a day. Some mild diuretic, such as citrate or acetate of potassium, is given. The pulse may be 80 or 90, and digitalis is therefore not given. Under this mild therapy the anasarca subsides, the albumin diminishes, and the urea and quantity of urine increase. Milk also tends to increase the quantity of urine. A bath at 104°–105° F. (40° C) is given every day or every second day according to the indications. The diaphoretic effects of vapor baths are less marked. In some of the severer cases the urine is greatly diminished, the anasarca extreme, the pulse and respirations are increased, and the temperature may be elevated. The anasarca is then treated by a daily warm bath, in which the patient remains for five minutes, and is then wrapped in a warm dry blanket to promote diaphoresis. A warm rectal enema at the temperature above mentioned is given twice daily. The kidneys are stimulated by means of digitalis and acetate, citrate, or tartrate of potassium. The digitalis is given in form of the infusion, ʒss–ʒj with 3 to 8 grains of the potassium salt, three or four times daily. The pulse is closely watched and not allowed to fall too low. The bowels are kept open by the daily administration of cathartics. If, as frequently happens, the heart becomes weak, sparteine or liq. ammoniæ acetatis and nitroglycerin may also be given. I do not administer preparations of musk or camphor in nephritis. Convulsions are best controlled by means of chloroform. Warm baths and high warm enemata are also useful. Bromide and chloral are also given by rectum, as in ordinary eclampsia.

In convalescence the question arises, When shall diuretics be discontinued? As soon as the quantity of urine is above the normal, they are of no further value. The baths and enemata are continued as long as there is the least œdema of the surface. Warm enemata should not be continued after the urine has increased to the normal amount. Ordinary enemata are then given for the purpose of aiding the cathartics in keeping the bowels open and clear of fecal accumulations.

Rest in bed should be continued until there is no palpable albumin reaction. Meat and vegetables are then added to the diet list. If anæmia is present, a readily assimilable form of iron, such as the peptonate, is given. Casts will appear in the urine far into convalescence. The patients may, however, be allowed to be up if they bear the change well. A too protracted stay in bed is sometimes exhausting in summer. If symptoms of anasarca and other signs of nephritis recur, the treatment is the same as in primary acute attacks. The treatment of chronic nephritis in children does not differ from that followed in the adult. I have recently subjected two children who suffered from the chronic diffuse form of nephritis following scarlet fever, accompanied by recurrent attacks of anasarca extending over years, to Edebohl's operation of splitting or extirpation of the kidney capsule. Both cases were much benefited by the operation. One case is now free from symptoms for fully a year. If we can improve these cases to this extent, the operation is certainly indicated, even if the operation is powerless to restore the kidney to the normal.

NEW GROWTHS OF THE KIDNEY.

Thirty-eight per cent. of all the reported cases of kidney tumors occurred in children (Döderlein, Lewi). The following growths are here considered: 1. Cysts of the kidney; 2. Tuberculosis of the kidney; 3. Carcinoma of the kidney; 4. Sarcoma of the kidney.

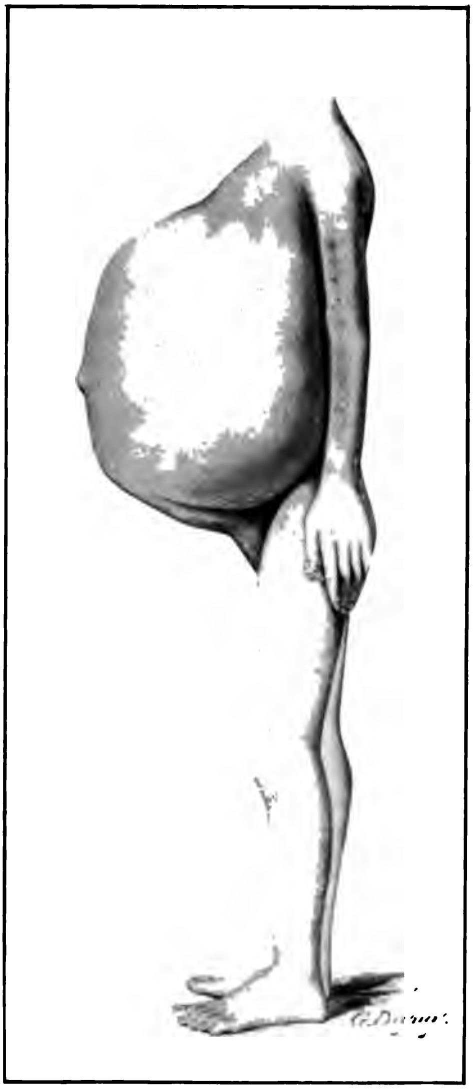
Cysts of the Kidney.

Cysts of the kidney are in children usually of congenital origin. They are formed in the second half of intra-uterine life. They are bilateral, only 1 in 60 being unilateral (Lejars). The kidney is made up of greater and smaller cysts. The cystic formations may be present to the entire exclusion of kidney tissue. The cysts may attain the size of a child's head and seriously obstruct delivery. They are of anatomical interest only, since infants with such cysts present other abnormalities and die soon after birth.

Hydronephrosis.

Hydronephrosis is either congenital or acquired. If acquired, it occurs late in childhood. The congenital form is due to stenosis in some part of the urinary tract. Hydronephrosis is as a rule unilateral. If it occurs after birth, it may be due to obstruction by calculi or to uric acid infarction of the kidney. The healthy kidney is physiologically enlarged. The acquired form is due to obstruction by calculi or to tumors pressing on the ureters. At first the pelvis of the kidney, then its tissue is encroached upon in the gradual dilatation. Finally the shape of the kidney is lost. There is a large fluctuating tumor which may or may not be painful.

PLATE XXVIII.



Sarcoma of the Kidney. Child six years of age
Irregular contour of the abdominal tumor.

When it is punctured, there is withdrawn a fluid which contains albumin, epithelium, urea, uric acid, and which has a low specific gravity. In some cases there occurs what is known as intermittent hydronephrosis. The contents of the tumor are emptied spontaneously, but reaccumulate. The diagnosis rests on the presence of a fluctuating tumor containing the fluid, and urine constituents. Cystoscopy may in some cases reveal obstruction of the ureters.

Cysts must anatomically be differentiated from the condition of hydronephrosis. Cysts are new growths (Senator); in that respect they differ from the cystic condition of hydronephrosis. It is not possible clinically to differentiate congenital cysts of the kidney from congenital hydronephrosis.

Sarcoma of the Kidney.

Sarcoma of the kidney occurs in children as a primary growth. In the statistics of Rosenstein and Senator two-thirds of the cases occur before the tenth year. It is more frequent in females. The left kidney is more commonly affected. Sarcoma occurs in the newly born infant. The presence of muscle, bone, and cartilage tissue in these growths supports the theory of their congenital origin (Jacobi). The anatomical nature of the growth varies widely. It may be round-celled or spindle-celled sarcoma, a fibro-sarcoma, myo-sarcoma, angio-sarcoma, melanotic sarcoma, or adeno-sarcoma. There may be metastases. The tumors sometimes attain a weight of fifteen pounds.

The **symptoms** do not differ materially from those of carcinoma of the kidney, nor is sarcoma of slower growth. In many cases the pain, hæmaturia, and tumor follow a traumatism. Hæmaturia is not, as in carcinoma of the kidney, a constant symptom. I have seen cases of both carcinoma and sarcoma of the kidney in young children without hæmaturia or growth elements in the urine. Ascites is present in more than one-half the cases (Lewi).

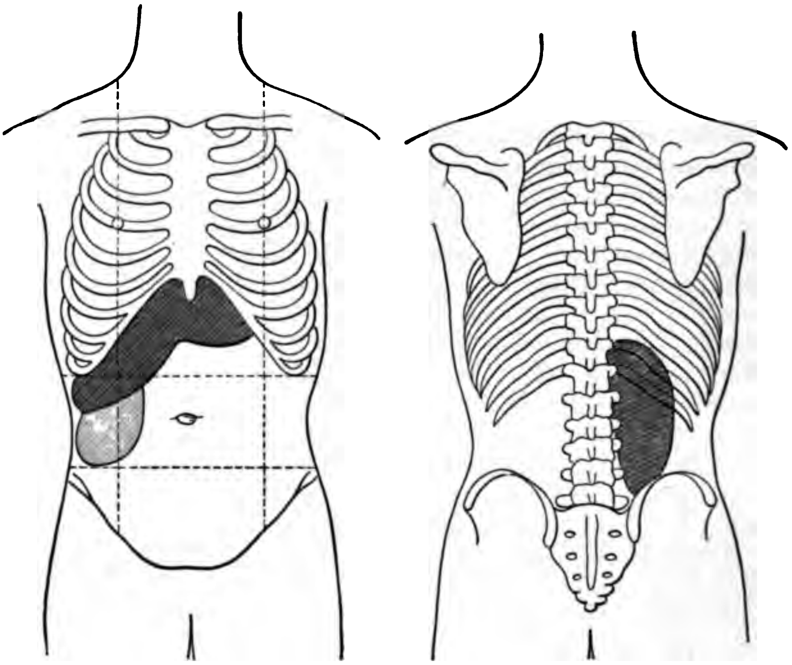
Diagnosis.—A malignant growth in a child may be surmised to be a sarcoma, since those growths are more frequent in children than carcinomata. Swelling of the lymph-nodes may be present in sarcoma as well as in carcinoma. Histological elements in the urine are rare. Von Jaksch has mentioned the presence of small round cells (sarcoma cells), but their significance is not as yet determined. Puncture for diagnostic purposes is dangerous, and if performed at all should be done posteriorly in the lumbar region (extraperitoneal). In sarcoma of the kidney, as in all growths of that organ, the colon is pushed in front of the growth (Plate XXVIII.).

Carcinoma of the Kidney.

Of 449 cases of carcinoma of the kidney (Rohrer, Ebstein, Lachman), 157, or almost 35 per cent., occurred in children under the tenth year. Monti tabulated 50 cases, and found that more than

50 per cent. occurred in children under the age of two years. The youngest patient was twelve months of age. It is more frequent in males. As a rule the right kidney is affected. In children, the growth is apt to attain great size. Guillet found that the average weight was eight and one-half pounds. By reason of the great weight of the growth, the kidney may sink from its normal position and lie transversely across the vertebral column. The growth is a primary one, The medullary carcinoma is the prevailing type; the scirrhous is next in order of frequency. The disease may be secondary to carcinoma of the suprarenal capsule or of the retroperitoneal glands. The liver, the lungs, and the inguinal lymph-nodes may be secondarily involved.

FIG. 163. FIG. 164.



Enlargements of the kidney.

Anterior palpable tumor beneath the liver.

Posterior area of flatness in the lumbar region, giving a palpable tumor between the border of the ribs and the crest of the ilium.

Symptoms.—The chief symptoms are pain, hæmaturia, cachexia, and enlargement of the kidney. Guillet found that hæmaturia was the first symptom in one-half the cases. The quantity of blood passed may be very small, or so great as to amount to a dangerous hemorrhage. The urine may be red or chocolate colored, and may contain clots of blood or casts of the ureters. Frequent micturition is sometimes an early symptom. In other cases there is no hæmaturia, the cachexia, emaciation, and tumor being the first symptoms. In younger children the hæmaturia is frequently absent. The kidney is in these cases protected from traumatism. The tumor is sometimes so great as to cause displacement of the organs. In Fürbringer's case the heart was displaced to a situation beneath the clavicle. The abdomen is distended, and the colon is pushed in front of the growth and is indicated by a tympanitic area at one side of the median line of the tumor. On the right side, the tumor appears beneath the liver, and in narcosis can be felt in that situation as a distinct mass. The tumor has an uneven surface. The urine may, in addition to blood, contain histological elements of the growth. This does not occur so frequently in carcinoma of the kidney as in tuberculosis of that organ.

Duration.—The progress of the growth is much more rapid in children than in adults. In the former subjects the duration of the disease is from ten weeks to fourteen months (Roberts).

Diagnosis.—In children, while the diagnosis of a morbid growth of the kidney can be made, it is not possible to differentiate between the symptoms of carcinoma and those of sarcoma. It cannot be determined, from the symptoms, whether the growth is a simple carcinoma, an adeno-carcinoma, or an adeno-sarcoma. The symptoms of a malignant growth of the kidney are pain, hæmaturia, tumor, and cachexia. A cyst of the kidney may be confounded with a malignant growth. Cysts are congenital, and as a rule bilateral. This is also the case in hydronephrosis. In the latter condition extraperitoneal puncture of the tumor may give a fluid with urine constituents. In carcinoma of the kidney, puncture for diagnostic purposes is not devoid of danger.

Tuberculosis of the Kidney.

Tuberculosis of the kidney is rarely if ever primary. Senator asserts that it never occurs as a primary lesion. There are pathologically two forms—the miliary and the cheesy. The miliary form is more frequent in children, the cheesy in later life. In the miliary form, the kidney tissue is the seat of an eruption of miliary tubercles. In the cheesy form, tuberculous nodules may entirely replace the substance of the organ. The cheesy form is as a rule secondary to tuberculosis of the genitals—the epididymis in boys and the tubes in girls. The symptoms do not differ materially from those of the same condition in adults. In the miliary form there are no symptoms. In the cheesy variety there are dysuria, strangury, vesical tension, pain in the region of the kidney, emaciation, and fever. The urine contains albumin, blood, epithelium, and pus cells, and is acid in reaction. Tubercle bacilli are sometimes found.

The **diagnosis** rests on the presence of tubercle bacilli in the urine, an enlarged palpable kidney, hæmaturia, and tuberculosis of other organs—the genitals or the lungs.

Treatment of New Growths of the Kidney.

The treatment of new growths of the kidney is within the province of the surgeon. The congenital cysts are of scientific interest only. If there is reason to believe that there is congenital hydronephrosis which is unilateral only, surgical interference is indicated. Sarcomata and carcinomata should be treated surgically if there is reason to believe that there are no metastases in the liver or elsewhere. Tuberculosis of the kidney is treated more from a general standpoint. If there is tuberculosis elsewhere, palliative treatment alone must suffice. Isolated tuberculosis of one kidney is a rare condition which necessitates extirpation of the organ. If it is impossible to determine the proper treatment, an exploratory operation is indicated.

Pyelitis—Pyelonephritis.

This is a very rare affection in infancy and childhood. Calculus, tuberculosis, and irritating drugs, are etiological factors. It occurs as a complication of the infectious diseases—scarlet fever, measles, variola, and typhoid fever. It may be caused by infection from a vulvovaginitis or by coli bacteria entering through obscure channels. The inflammation of the pelvis of the kidney may extend to the tissue of the kidney itself. Small abscesses are present in the cortex and there is degeneration of the parenchyma of the kidney.

The **symptoms** consist of fever, of an intermittent or remittent type, with recurrent rigors. The fever may alternate with subnormal temperatures. The urine is characteristic. It is of normal quantity, and low specific weight, is acid in reaction, milky in appearance, and contains pus, mucus, and albumin. Microscopically there are casts, leucocytes, and bacteria. The bacteria present in recently voided urine include the coli group, Bacterium lactis, proteus, and Bacillus pyocyaneus (Baginsky). It is characteristic of this condition that the pus may suddenly disappear from the urine through occlusion of the ureter on the affected side by calculi, fibrin, or inspissated mucus. It may reappear after a time. Emaciation and periarticular or intermuscular inflammation are also sometimes present.

Prognosis is influenced by the causal agent. Simple coli-pyelitis may retrograde within a few weeks and result in recovery. The chronic form may last for months. Other forms may lead to irreparable inflammation and degeneration of the kidney.

Treatment.—If the condition does not improve under treatment of rest, milk diet, and diuretics, surgical interference may become necessary.

PERINEPHRITIS AND PARANEPHRITIS.

This condition is rare in infancy and childhood. It is not always possible to determine the cause. If such is the case, the disease is called primary. As a rule, it is secondary to traumatism in the lumbar region, to pyelitis, or to pyelonephritis. It may occur in septicopyæmic processes, and I have seen it follow the infectious diseases, notably scarlet fever. Of 166 cases collected by Nieden, only 26 occurred in children. One case occurred in an infant five weeks old. Gibney's cases ranged from one and a half to ten years of age. The condition is more common on the left side. The pus may burrow behind the liver or spleen, or find its way downward, forming a mass simulating a cold abscess or a perityphlitic abscess. It may perforate into the pelvis of the kidney, the intestine, peritoneum, vagina, or diaphragm, or, may pass along the ileopsoas muscle, and find its way to the hip, and thus appear externally. The kidney may be involved because of its contiguity to the seat of the process. Pleuritic metastases and amyloid degeneration may finally result.

The **symptoms** are usually obscure. The fever is intermittent or remittent. Young children do not as a rule complain of pain. The first intimation of the nature of the disease is the appearance of a swelling in the lumbar region. On bimanual palpation, a tumor which is fixed, tense, and does not move with respiration, is felt deep under the liver, in the region of the cæcum and ascending colon on the right side, or underneath the spleen on the left. Gibney has described these cases and shown how they may be easily mistaken for cases of cold abscess. The thigh of the affected side is held in a condition of semiflexion.

The **treatment** is surgical.

ENURESIS NOCTURNA AND DIURNA.

This is a functional neurosis of the bladder in which the urine is passed involuntarily, and, as a rule, at night during the first hours of sleep. It may, however, be passed at any time during the night. Some patients have at times no control over the bladder during the day (diurna). Some have enuresis every other night or only once or twice a week, and others suffer from the affection every night.

Cases of enuresis should be differentiated from those in which there is a complete paresis of the sphincter vesicæ. In the latter case the urine simply flows away. These are cases of disease or anomaly of the cord (spina bifida). In enuresis the children may in other respects be in good health. There is frequently a nervous condition. In some cases there is lithiasis or stone in the bladder; in others the etiological factor is Oxyuris vermicularis, obstipation, tumor of the bladder, or vulvovaginitis. Cystitis and adenoids have been regarded as causal. In the majority of cases no cause can be found. The condition follows the exanthemata. In boys it usually disappears toward the sixteenth year. I have seen it persist in girls into adult life. Its treatment becomes a very serious problem.

The **diagnosis** is not difficult. The urine should be carefully examined for evidences of lithiasis, cystitis, glycosuria, nephritis, and nematodes, and the bladder for stone. The diagnosis is not made in infants and very young children. In the latter the enuresis is often only apparent. They do not know how to indicate their wants.

Treatment.—The urine should be passed before retiring. The patients should take little liquid at the evening meal. The foot of the bed is raised so that the head is slightly lower than the pelvis. The drugs most utilized are ergot and atropine. The former is given in the fluid extract, minims x to xxx (0.6 to 2.0) t. i. d. Atropine is given before retiring in a solution (grain j to ℥ij; 0.06 to 30.), a drop for every year of the age (Watson). It is efficient in many cases, but in some children distinctly dangerous. I had one case in which I gave one-half the above dose. The child, five years of age, became slightly delirious and tried to walk out of a window. Many cases will improve, only to be subject to relapses. Marion Sims has shown that enuresis in young girls may be due to an intolerant and very small, contracted bladder. In such cases, he advises gradual dilatation of the bladder by injecting the organ with increasing quantities of an indifferent fluid. If treated in this way, the bladder will eventually retain urine. Most of the cases resist all methods of treatment.

VULVOVAGINITIS.

(*Urogenital Blennorrhœa.*)

The term vulvovaginitis, or, as it is now called, urogenital blennorrhœa, refers to a gonorrhœal inflammation of the genital tract in children. Before describing the condition it is necessary to refer to catarrhal conditions which are not gonorrhœal, and which are present in the normal state.

Epstein has shown that in the newly born infant there is a physiological and normal discharge from the vagina. It is an adhesive, mucoid discharge containing epithelial cells and micro-organisms. A few days after birth, this discharge assumes a purulent and, in icterus, an icteric hue. No leucocytes are found in the discharge. In two weeks it ceases and the parts appear normal. This form is not gonorrhœal. A second condition which I have noted in very young children is the result of uncleanliness, lithiasis, irritation caused by Oxyuris vermicularis, or masturbation. The parts are reddened and eroded, and are bathed with an abnormal serous discharge. There may be a few erosions around the introitus. These cases recover with ordinary care and removal of the source of irritation. Pus is rarely secreted.

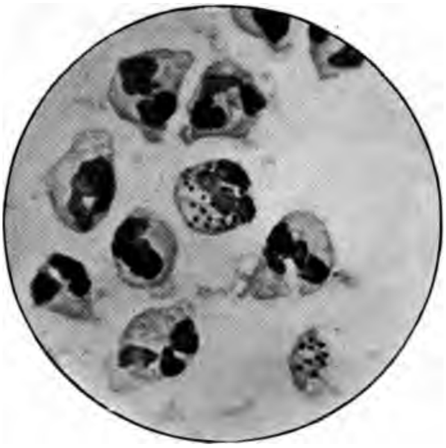
A second group of cases occurring in young female children includes those of vulvovaginitis of the simple catarrhal type. These have a scanty or profuse purulent discharge from the vagina, vulva, and urethra, which presents clinically all the features of the specific gonorrhœal group, but is not gonorrhœal. The condition is not of infrequent occurrence. The urethral orifice is swollen and red. The hymen is also swollen and inflamed. The discharge is thin and milky, or greenish and viscid. Microscopically, it shows in the pus-cells bacteria and diplococci in groups, but these do not show either by culture or on staining the characteristics of the gonococci. The history of such discharges is singularly similar to that of the gonorrhœal form. Urination is painful, and the discharge persists even under careful treatment. In one case of this kind I have seen an inguinal bubo. The catarrh, like the gonorrhœal form, affects the urethra, vulva, vagina, and cervix uteri. I am convinced that the discharge is infectious and communicable from one child to another. It may last for months and again recur. Its exact etiology is still unknown. Uncleanliness, infection from a vaginal discharge, marasmus, the infectious diseases, or frail health may be the cause.

Urogenital Blennorrhœa.—These cases have been described by Pott, van Dusch, Spaeth, Cahen-Brach, Epstein, and others.

Etiology.—This form may occur in newly born infants (Epstein) or in older infants and children. Epidemics may occur in hospitals (Fränkel). The avenue through which the disease is conveyed is still unknown. It occurs in all walks of life. In some cases there is a history of the child's having slept with the mother. In others, there is no such history. I have sometimes obtained a history of an abnormal attempt at coitus between boys and girls, the boys having suffered at the time from gonorrhœa. Such cases are, however, exceptional. The exciting cause is the gonococcus (Neisser) (Fig. 165). This micro-organism has been found in the discharges of all these cases, and cultivated (Koplik, Heiman).

Symptoms.—There is a thick, viscid, purulent, greenish or yellowish discharge from the vagina, which bathes the parts and dries in crusts on the labia. The opening of the urethra is reddened and swollen. There is a discharge from the urethra. Micturition is painful. In some cases there are slight swellings of the inguinal lymph-nodes. If the speculum which is used for the male urethra is introduced into the vagina (Tuttle's urethral speculum), it is seen that the purulent discharge is present in the folds of the mucous membrane of the vagina. The cervix uteri also contains a drop of pus. Thus the whole genital tract is involved. Some children complain of pain over the lower part of the abdomen. On examination, this is found to be pelvic, and is probably due to inflammatory reaction of the tissues about the uterus and vagina.

FIG. 165.



Gonococci in vaginal discharge. Cover-glass spread. Photomicrograph. × 1000.

Complications and Course.—The course of the disease is quite tedious, and may occupy eight weeks, three months, or more. The discharge may abate, only to return in its original severity.

Peritonitis has in rare cases been reported as a complication of this form of vulvovaginitis. It may prove fatal. Hunner and Harris recently reported a fatal case in a girl ten years of age. They collected 5 other cases from the literature occurring in children. Pelvic peritonitis occurred in 2 of my cases with the usual signs of pain and fever. Both cases made a good recovery.

Hartley and I have reported cases of arthritis complicating vulvovaginitis in children. My cases occurred in the first and second weeks of the disease. In one case, only one joint was affected; in another, two. Both recovered without suppuration.

Gonorrhœal conjunctivitis may result from careless infection of the eyes. I have had only 2 cases in which the patients complained of præcordial pain. In neither were there active symptoms of endopericarditis, but there is no reason why it might not occur in children, as in adults.

Sänger at one time traced a connection between sterility in later life and attacks of this disease in childhood.

Treatment.—Prophylaxis is of great importance. A child affected with the disease should not be allowed to sleep with other children. The toilet appliances should not be used by other children. The parents should be carefully enlightened concerning the infectious nature of the affection and the great danger to the eyesight should infection of the eyes occur. The hands of the patient should be kept scrupulously clean. In institutions the patients should be strictly isolated. The vulva should be kept covered with a pad of absorbent gauze, and a diaper should be worn over this to prevent the discharge from soiling the clothes. In the acute stage, the vagina should be irrigated with a glass catheter or a Skene urethral catheter twice daily. The solution should be at a temperature of 108° F. (42.2° C.). The irrigating solutions should be either a 2 per cent. solution of acetate of aluminum or a 1 : 2000 or a 1 : 500 solution of nitrate of silver. If the silver or aluminum solution is irritating, a simple saturated solution of boric acid may be used. In the subacute stage the vagina is painted every other day with a 5 or 10 per cent. solution of nitrate of silver. A Tuttle urethral speculum is used for the purpose. If the child is intractable, it is impossible to do this without the use of an anæsthetic, which, however, seems scarcely justifiable. I have cured these cases with rest in bed and irrigations. I have tried the bougie treatment and the protargol and permanganate of potassium irrigations, but have found the treatment above described preferable.

URETHRITIS IN MALE CHILDREN.

Simple urethritis of the anterior portion of the urethra occurs in infants and young children. It is caused either by unnatural interference with the parts or infection. It is not gonorrhœal. The meatus is slightly red or the parts are agglutinated with dried pus. On pressure, a drop of pus exudes from the urethra. There is ardor urinæ, due to a slight fissuration of the meatus. The affection is easily cured by attention to cleanliness. An alkali, such as citrate of potassium, is given in very small doses, to alleviate the ardor urinæ.

Gonorrhœa occurs in male infants and boys, and is the result of direct infection. The symptoms are much the same as in adults, except that, as a rule, there are no complications. Balanoposthitis and lymphadenitis may occur, also epididymitis, and rarely orchitis. Bokai reports cases of stricture.

CYSTITIS.

Cystitis is not very common in infants and children. Attention has recently been drawn to this affection by Escherich, who reported several cases of cystitis in young female children caused by coli bacteria.

Etiology.—Barlow classifies cases of cystitis as follows:

(*a*) Chemical cases, caused by drugs.

(*b*) Bacillogenous cases, caused by the tubercle bacillus, the Urobacillus liquefaciens, Bacillus coli communis, and the typhoid bacillus.

(*c*) Coccogenous cases, caused by the gonococcus, staphylococcus, streptococcus, and various diplococci.

Of all the micro-organisms mentioned, the Bacillus coli communis is the most frequent cause of cystitis (Melchior).

The direct exciting causes of cystitis in children, as in the adult, are cold, catheterization, or calculi. It may follow urethritis, vulvitis, or may complicate the infectious diseases—scarlet fever, typhoid fever, and diphtheria. The changes in the bladder are, as in the adult, swelling and hyperæmia of the mucous membrane. In chronic cases there are thickening of the rugæ, ulcerations, hemorrhages, and the formation of false membrane (diphtheria).

Symptoms.—The symptoms are deep-seated pelvic pain, a desire to pass urine, and, frequently, pain in urination. There may be slight fever. The urine is passed in small quantities, is cloudy, and contains flocculi and shreds of mucus and muco-pus. There is sometimes a sediment of creamy consistency. The urine may contain blood and pieces of false membrane (diphtheria). In tuberculous cases, there are tubercle bacilli in the urine.

The cases may be acute, lasting only a week or two, or may be chronic, and last for months. In the variety caused by the coli bacteria (Escherich) the urine is acid. It may be acid in other acute forms. In the chronic tuberculous cases the urine may be alkaline and contain crystals of triple phosphates. The tendency in acute cases is toward recovery. Complications, such as pyelitis and peritonitis, may occur.

Treatment.—If the symptoms are acute, the child is put to bed, and the bladder washed out with a solution of creolin. Salol, grain ij (0.12), is given three or four times daily. Urotropin, grain iij (0.18), is of great utility in the ammoniacal forms of cystitis. Alkaline waters (Vichy) are given freely and the bowels are kept open with alkaline salts. In convalescence an alkali, such as citrate of potassium, given in grains v (0.3) t. i. d., is beneficial.

In chronic forms saccharin is sometimes the only drug that will give relief. Grains ij (0.12) t. i. d. may be administered to a child of six years with safety.

Bacteriuria.

This is a condition in which the urine assumes a very fœtid odor, as of decomposition, becomes filled with bacteria, generally of the coli or the Staphylococcus albus group; the urine is usually acid in reaction, though cases are recorded in which the reaction was alkaline or neutral. There are no evidences of cystitis or inflammation of the urogenital tract.

Occurrence.—It occurs, as a rule, in delicate children. Most of the cases coming under my observation were suffering from various affections of the gastro-enteric tract. Female children are mostly affected. It occurs in children from the fifth to the thirteenth year of life.

Symptoms.—Some cases set in with fever, headache, vomiting, and pain over the bladder. Other children complain of dysuria; whereas in many cases the onset is insidious, and when consulted the physician encounters the following symptoms: If old enough to describe their symptoms, the little patients complain of slight headache, and there is a slight febrile reaction. The main symptoms are confined to the urine, which is passed frequently, with pain referred to the bladder. The urine is dark, has a musty or foul odor, and is acid in reaction as a rule. It contains no albumin, or only a trace; no sugar; and there are present no evidences of cystitis. There is no inflammation of the genitals. In most cases the Bacillus coli communis is found in pure culture; in other cases, the Staphylococcus pyogenes albus is present.

Pathogeny.—The manner in which the urine is infected in these cases is still a matter of discussion, though many are agreed that the affection may occur in the majority of cases by way of the gastro-intestinal tract, or in younger children by way of the urethra.

Treatment.—The prognosis is good in most cases, inasmuch as recovery occurs in periods of from one to several weeks.

The children should be kept quiet in bed. Vichy water and salol are administered internally; the bladder is washed with a 3 per cent. solution of boric acid in severe cases, or ½ per cent. solution of lysol.

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SECTION XIII.

DISEASES OF THE NERVOUS SYSTEM.

METHODS OF DIAGNOSIS.

Lumbar Puncture.

LUMBAR PUNCTURE was first practised by Quincke. It is to-day one of the most useful adjuncts to the methods of diagnosis in acute and chronic forms of cerebral and spinal disease. Its future usefulness as a therapeutic measure is not clearly established, but will probably lie in relieving symptoms due to pressure, and removing the excess of inflammatory exudate in the various forms of meningitis.

The Normal Cerebrospinal Fluid.

Normal cerebrospinal fluid is a clear colorless fluid having a slightly alkaline or neutral reaction. Its specific gravity varies from 1007 to 1009. It contains from 0.05 to 0.1 per cent. of albumin (Quincke, Rieken, Pfaundler), and because of the presence of sugar has a slightly reducing action on copper. It does not coagulate spontaneously. If centrifuged, a microscopic sediment of a few endothelial cells and leucocytes may be obtained. The cerebrospinal fluid is normally under a pressure of from 5 to 35 millimetres of mercury. The pressure in infants is lower than that in children. The causes of the variations of pressure and the nature of the conditions under which they occur have not as yet been determined. Respiration causes a deviation of fully 6 millimetres of mercury in the manometer column.

Abnormal Conditions.—The cerebrospinal fluid will in pathological states vary in respect to specific gravity, composition, appearance, and in the amount of sediment contained. The pressure in the subarachnoid and cerebrospinal spaces will also vary in different forms of disease.

The **specific gravity** in tuberculous meningitis varies from 1003 to 1011 (Lenhartz), in cerebrospinal meningitis from 1005 to 1012 (Pfaundler).

The **gross appearances** of the fluid obtained by lumbar puncture may be changed by the admixture of blood. Blood may come from the puncture wound or may have been in the canal previous to puncture as a result of a hemorrhagic pachymeningitis or of some form of cerebrospinal meningitis, traumatism, or apoplexy with rupture into the ventricles. The wounding of veins either in the tissues or in the cauda equina may cause the admixture of blood. The quantity of blood may be just sufficient to tinge the fluid or the blood may be almost pure. It is not possible to determine whether the admixture of blood is or is not the result of accidental puncture of a vessel unless, as in pachymeningitis or traumatism, light is thrown on the matter by the history of the case and the presence of blood on repeated puncture. The accidental admixture of blood is unfortunate, since it obscures the microscopical diagnosis. The hemorrhage into the spinal canal is never alarming or of serious import.

Tuberculous meningitis changes the gross appearance of the fluid obtained by lumbar puncture. The fluid may be quite clear, exceptionally cloudy, opalescent, or in rare cases purulent. As a rule, however, it is clear in the early stages of the disease and cloudy in the later period. If the test-tube is held in a strong light, there may be seen, in a clear or cloudy fluid, myriads of highly refracting particles resembling the motes in a sunbeam (Moser, Bernheim, Pfaundler). The appearance is quite characteristic. It was first explained by Lichtheim, as the result of spontaneous coagulation. If a test-tube of the fluid obtained by lumbar puncture is placed in the upright position in an ice-box, there is found after twenty-four hours, a fully formed cobweb-like, funnel-shaped coagulum, beginning a little below the surface of the fluid and extending downward, the broader part of the funnel being above. According to Pfaundler, this coagulum is of diagnostic import. I have relied on its appearance in fluid which was not contaminated with blood, and found it of great value. The formation of the coagulum begins after the fluid has stood for two hours, and is fully completed by the following day. It is usually found from eight to twelve days before death.

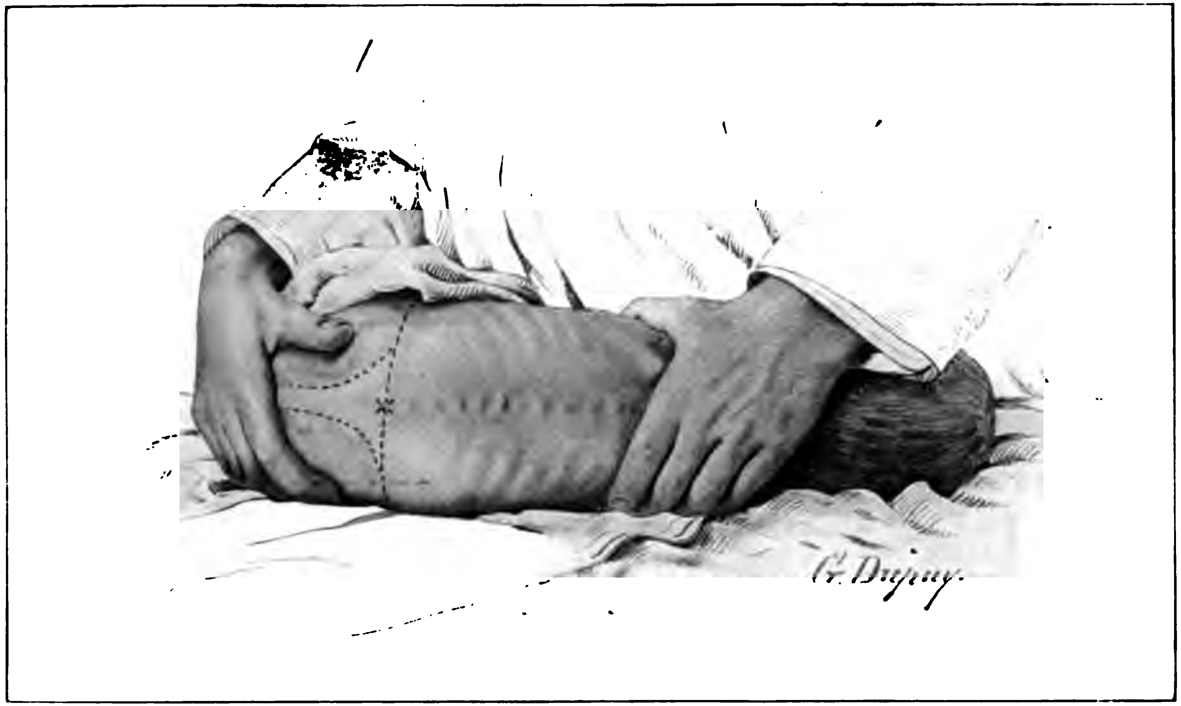
Suppurative Meningitis.—In this form of meningitis, the fluid obtained by lumbar puncture is purulent, opalescent, grayish-white, grayish-yellow, or brownish (hemorrhagic). Exceptional cases give a clear fluid. There may be a spontaneous coagulum resembling that seen in tuberculous meningitis.

Epidemic and Sporadic Cerebrospinal Meningitis.—In the early stage of this disease, the fluid may be quite clear with suspended microscopic sediment. It may also be cloudy or thick, creamy or bloody. It may at first be clear, and later in the disease become purulent (Councilman).

Chronic Hydrocephalus.—This gives a clear fluid with no suspended particles visible to the eye, although microscopially there may be leucocytes. Pfaundler in one of his cases obtained a fluid which was cloudy because of the admixture of leucocytes.

Tumor of the brain gives a clear fluid. I have had a case of this kind.

PLATE XXIX.



Operation of Lumbar Puncture. Method of holding the patient. Dotted lines show topography of the parts and the manner of finding the proper point for puncture.

Sediment.—This feature will be fully discussed under the sections devoted to Tuberculous Meningitis and Cerebrospinal Meningitis.

The **pressure** under which the cerebrospinal fluid is retained in the subarachnoid space and in the spinal canal is increased in the various forms of meningitis. This is especially true of tuberculous meningitis, in which the pressure may reach 110 m.m. of mercury. In this disease the pressure increases from the initial period to that of pressure symptoms, and diminishes toward the close of the disease—the stage of paralysis. Ventricular involvement gives the highest pressure figures. The following figures are taken from Pfaundler's tables:

First stage	48 m.m. of mercury.
Stage of pressure	52 m.m. " "
Stage of paralysis	24 m.m. " "

In suppurative meningitis, the pressure varies from 10 to 37 m.m. of mercury; in cerebrospinal meningitis, from 24 to 50 m.m.; in hydrocephalus, from 6 to 60 m.m.; in tumor of the brain, from 3 to 52 m.m. (Quincke, Slawyk, Pfaundler).

The presence of an increased amount of albumin in pathological states has been noted by Wentworth, Quincke, and Pfaundler. In tuberculous meningitis it may reach 0.3 per cent.; in purulent meningitis, 0.6 per cent.

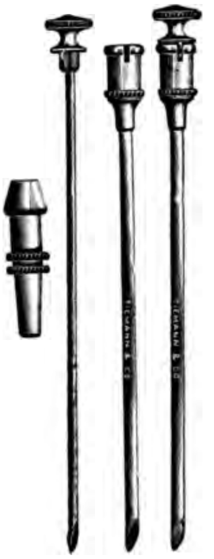
The Operation of Lumbar Puncture.

The **instrument** consists of a trocar and canula such as is employed in tapping cavities. The best form of instrument is that devised by Quincke (Fig. 166). The canula should be at least one millimetre in diameter. It is not necessary to use a manometer. In infants the tenseness of the fontanelle is a rough guide in estimating the pressure in the subarachnoid space.

Place of Puncture.—The puncture is made in the space between the third and fourth or the fourth and fifth lumbar vertebræ. This point is obtained by palpating the crests of the ilium; an imaginary tangent to these crests strikes the fourth space. The space above this imaginary line will, as a rule, be found to be the third space. Puncturing the canal in the space between the sacrum and coccyx or in the lower sacral space offers no advantages either anatomically or from a diagnostic standpoint.

Method.—Local anæsthesia only is necessary. The back of the patient is carefully scrubbed with green soap, then washed with alcohol and ether, and finally with sublimate. The patient is laid on either side according to the convenience of the operator. The spine is curved so that the spinous processes may be distinctly seen and palpated (Plate XXIX.). No considerable pressure should be brought to bear on the neck, since in cerebrospinal meningitis or in the basilar form of meningitis in which there is opisthotonos, serious injury to the neck may be caused. The spine is curved from the shoulders and pelvis. The needle, having been previously boiled, is introduced in the median line between the spinous processes and is directed upward (Plate XXX.). When it is in the canal, it is perceived that there is a lack of resistance, and that the point of the instrument is free. The canula is withdrawn and the first drops caught in a sterilized test-tube. A second test-tube is substituted for the first after a few drops of bloody fluid have been allowed to flow out, and from 10 to 50 c.c. of fluid are withdrawn, the amount varying with the pressure. If it flows drop by drop, 20 c.c. are sufficient for diagnostic purposes and also to relieve the pressure. If there is opisthotonos and the fluid does not flow well at first, cautious straightening of the neck will facilitate the outflow. In infants the fontanelle is a good guide in gauging the pressure. As soon as a few cubic centimetres of the fluid have been withdrawn, the fontanelle will be felt to be considerably flattened and relaxed or even depressed. Heubner has withdrawn 100 c.c., but the removal of such large quantities is unnecessary and may be followed by hyperpyrexia and collapse. I rarely withdraw more than 20 c.c. If there is a dry tap, the canula should be withdrawn and a second attempt made on the following day. A dry tap may be caused by a fibrin clot or by the falling of the cauda equina in front of the opening of the canula. The fluid may be viscid and refuse to flow. In that case the fluid should not be aspirated with a syringe, since in the experimental laboratory this method has been proved to be hazardous. If carried out as above directed, I have never seen any ill results from the operation of lumbar puncture. After puncture, the canula is rapidly withdrawn and the wound dressed with iodoformized gauze.

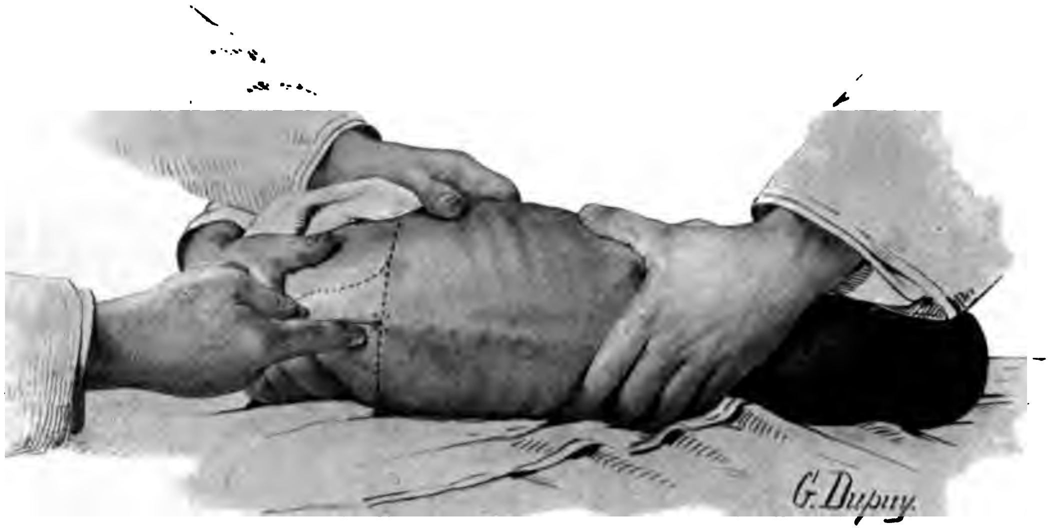
FIG. 166



The Quincke needle for lumbar puncture.

Indications for Lumbar Puncture.—Lumbar puncture is performed in the various forms of meningitis for diagnostic purposes, to determine the character of the fluid. It is not always an easy task to decide whether it should be resorted to. The decision is especially difficult in private practice, where the procedure is regarded with dread. Any marked disease of the lungs should first be excluded. In many cases of pneumonia the cerebral symptoms are marked. Only very marked symptoms of cerebral press-

PLATE XXX.



Operation of Lumbar Puncture. Method of introducing the puncture needle.

ure and the suspicion of pneumococcus meningitis should cause the physician to resort to puncture in order to fix the diagnosis. It is best not to perform it while the lesion in the lung is markedly active. Cases of tumor of the brain should not be subjected to puncture.

Indefinite cerebral symptoms, such as headache, restlessness, and convulsions of a general character, are not indications for puncture. I have seen cases of meningitis of the cerebrospinal type which gave few symptoms of disease, there being indefinite sopor, general muscular weakness with delayed reflex at the knee and marked emaciation, but no marked rigidity of the neck.

In doubtful cases I refrain from puncture. Cases with meningeal symptoms, in which there is a history of a blow, are proper subjects for puncture, since it is necessary to differentiate between meningitis, and abscess of the brain. In the various forms of purulent meningitis, symptoms of pressure such as convulsions, and signs of suppuration such as chills, are indications for puncture. After the first puncture is made and the diagnosis fixed, I do not in tuberculous meningitis repeat the operation.

The indications for puncture in other diseases such as hydrocephalus will be discussed under the headings of the various affections.

CONVULSIONS IN INFANCY AND CHILDHOOD.

Eclampsia Infantum.

Convulsions are a series of violent clonic contractions of a number of muscles, or of the muscles supplying one limb. There is always more or less of a tonic spasm at first. The convulsions are paroxysmal and accompanied by a loss of consciousness. In this section the acute convulsions of infancy and childhood are especially considered, and will be differentiated from certain spasmodic affections, such as laryngismus, tetanus, and epilepsy, which are accompanied by spasms, though classed by some as forms of convulsions.

Classification.—Convulsions of infancy may be classified as those which are primary or idiopathic, and those which are secondary, reflex, or symptomatic. In the first rubric are included the convulsions which occur spontaneously, or after some sensory irritation, very often of an obscure origin, such as epileptic, hystero-epileptic seizures, and tic. With increasing knowledge this class is gradually becoming more and more limited.

In the second class the symptomatic or reflex convulsions are included: (*a*) the cases which follow abnormal conditions of the circulation in the brain, such as anæmia or hyperæmia; (*b*) convulsions which occur at the outset of infectious diseases; (*c*) convulsions which are caused by disturbance of metabolism, and which occur at the outset or in the course of certain diseases in which toxins are thrown into the blood; (*d*) those which follow some peripheral irritation, such as occurs in a reflex manner in wounds, burns, etc., or directly reflex, as in meningitis, tumors of the brain, hydrocephalus, brain compression, poisons circulating in the blood (lead).

Occurrence.—The acute convulsions of infancy and childhood are symptomatic, and occur chiefly during the first half year of life. Fully four-fifths of the cases occur before the end of the second year of life. They are uncommon after this period; but a child who has had convulsions of the symptomatic type in infancy is likely to have a recurrence of the convulsions up to the seventh year of childhood.

Etiology.—The occurrence of convulsions necessitates not only the presence of an exciting agent or irritating substance, but there must exist a certain constitutional disposition or predisposition to convulsions, which may be hereditary. Soltmann has shown that in the newborn animal irritability of the motor nerves is almost nil, and that of the sensory nerves much below what is attained in later life. In the newborn, also, there is an absence of reflex inhibition, and the brain lacks volition; in other words, there is an absence of the psycho-motor centres. The inhibitory centres do not develop in parallel lines with the peripheral irritability of the sensory nerves. Reflex irritability is very much diminished at the outset, but increases later, becoming, at a certain period of infancy, above what is found in the adult. The musculature of the infant, on account of the instability of the nervous centres, can be thrown into tetanic contraction by the least irritation. This period of increased reflex irritability of the nervous centres has been placed by Soltmann at from the fifth to the eleventh month of infancy, thus corresponding with what is found in the human subject clinically.

Although the theories of Soltmann are not wholly endorsed by other observers, it remains true that in infancy the inhibitory centres are not fully active, that the psychomotor centres are absent, and that this is a period of increased reflex irritability of the peripheral nerves. In a causal sense, not only does this increased reflex irritability predispose to acute convulsions in infancy and childhood, but with it there is a hæmatogenous toxic element especially active at this period of life.

In infancy we have also the hereditary predisposition to neuroses, and tendencies derived from neurasthenic, alcoholic, syphilitic, and tuberculous parents.

It seems, therefore, that causal agents of acute convulsions in infancy and childhood are principally periodical toxins, such as are present in the circulation (hæmatogenous) at the outset of infectious diseases, as acute amygdalitis, exanthemata, typhoid fever, malaria, influenza, pertussis, mumps—all of which may be ushered in with a convulsion.

The explosion appears to be caused by the initial effect of the toxins and temperature on the ganglion-cell. Convulsions sometimes takes the place of the initial chill in pneumonia and malarial fever.

The disturbances of metabolism which may cause toxins to be thrown into the circulation occur in connection with gastro-enteric disease of any kind or with indiscretions in diet. Children who eat an excessive quantity of meat are particularly subject to these seizures. In addition to the above exciting agents we have mentioned also disturbances of the circulation which may cause convulsions, and these are found in connection with pertussis, bronchitis, and heart disease. In these affections there is an accumulation of carbonic acid gas in the circulation, which is the exciting agent of the intitial explosion; and, finally, we have as causes of convulsions the direct effect of mineral poisons, such as lead, circulating in the blood.

Convulsions, according to some authors, may be caused by the presence of alcohol in the mother's milk. This is a very questionable cause of convulsions. Rarely, convulsions may be caused by reflex irritation of a foreign body in the stomach, or by overdistention of the stomach during stomach-washing, an instance of which the author has seen; by burns, wounds, effects of cold, incarceration of a hernia. Retention of urine may, by reflex peripheral irritation, cause convulsions. The toxic form of convulsions occurs in uræmia.

Dentition is frequently mentioned among the causes of convulsions. Since dentition in a normal infant is devoid of symptoms, it is straining a theory to ascribe convulsions to irritation of the trigeminal branches. The acceptation of this dentition theory might lead one to overlook some serious condition, of which the first indication is an eclamptic seizure.

Under the heading of circulatory disturbances might further be mentioned an acute cerebral anæmia, caused by severe hemorrhage, which may give rise to a convulsion. Such convulsions are hardly included under the conception of infantile convulsions of the acute type.

The **pathogeny** of convulsions in infancy and childhood is the same as in the adult. The explosions are due to irritation of the centres in the ponto-bulbar junction, or in the area of Rolando (Hughlings Jackson). The starting-point of every convulsion is a ganglion-cell. It is not known whether the inherited neurotic tendencies already mentioned are powerful factors during infancy, or whether alcoholism or epilepsy in the family are active in causing convulsions of the purely acute type in infancy. Rachitic children, however, according to Kassowitz and Elsässer, are peculiarly subject to convulsions, because the cranial bones are the seat of hyperæmia and softening. The motor areas adjacent to these points of hyperæmia and softening are supposed to be in a state of constant irritability.

Kussmaul and Tenner have demonstrated that there is an acute anæmia of the brain during convulsions. On the other hand, it often happens that the convulsion is the cause of the bursting of a cerebral vessel. In such cases the signs of cerebral surface hemorrhage are present at autopsy. In other cases, although death has occurred during a convulsion, nothing is found post mortem but an œdema of the brain substance, of doubtful origin.

Symptoms.—The majority of convulsive seizures in infants and children are single. In certain cases the convulsions are repeated and extend over a prolonged period. The latter are not cases of simple acute infantile convulsions. The symptoms of acute eclampsia are sometimes so very slight as to be scarcely noticeable. A very observant mother will see a slight twitching of the lips and eyelids, a momentary turning of the eye and cessation of breathing, or a momentary spasm of the whole trunk. The expression "internal convulsion," so frequently heard, evidently denotes these slight eclamptic seizures. The genuine convulsion comes on without premonitory symptoms. There is a momentary spasm of the body, the head turns to one side and upward, and there is a corresponding upward direction of the eye. Then follow a series of clonic spasms involving the upper and lower extremities, and lasting for some time.

The hands are clenched, the forearms flexed, the body rigid, the lower extremities extended, the head thrown back. This tonic, momentary spasm is followed by a clonic spasm, beginning in the muscles of the face and involving those of the trunk and extremities. The teeth are set, the tongue is protruded and may be bitten. There are cyanosis and frothing at the mouth. The respirations are short and hissing, the pulse is imperceptible, and at the outset of the convulsion the heart becomes slow and irregular. A cold perspiration bathes the surface. The convulsive seizure may be momentary, may last a few minutes to a quarter of an hour, or one spasm may be followed rapidly by others extending over the same period of time. Toward the termination of the convulsive spasm the clonic contractions become less frequent; the child passes into a sleep or coma. In some cases the clonic spasms may be limited to one side of the body.

The child may be in a state of eclampsia for an hour, after which it may pass into the comatose state. The coma may be momentary or may merge into a sleep of variable duration. The end of the convulsive spasm is signalized by muscular clonic spasms decreasing in severity, until finally a long-drawn inspiration ends the attack.

Diagnosis.—It is very important to be able to distinguish between the various forms of convulsive seizures. Those occurring immediately after or within a few hours or days of birth have a different etiology from those just described. They may be caused by cerebral hemorrhage, and there will be symptoms after the convulsions, such as palsies, contracture, difficulty in deglutition, and prolonged coma. In these cases the convulsions are repeated. Atelectasis of the congenital variety may cause convulsions. The patients have slight or marked cyanosis, and, in the intervals, increase of respirations and signs of bronchitis and collapse of the lung.

Tumor and abscess of the brain, and meningitis, both cerebrospinal and tuberculous, may be ushered in by convulsions. In tumor, the convulsions are limited to the area in which the tumor or abscess is localized. In forms of meningitis, there will be the symptoms of that disease. Drugs and poisons may give rise to convulsions. The history of such cases will be of service. Cases of tetany and tetanus have convulsions in the course of the disease. In tetany there may be several convulsions in the course of twenty-four hours. Tonic spasm is the chief feature of the convulsion in tetany and tetanus. The clonic form distinguishes acute convulsions. In tetanus there is slowly increasing opisthotonos. In tetany the body may be lax in the interval, but there are rare cases of tetany which resemble tetanus in that there is rigidity in the intervals between the spasms. In tetany the extremities have a characteristic position. In some cases of simple acute infantile convulsions, an increased irritability of the nerves and muscles to mechanical stimulus remains for days after the paroxysms. The Chvostek and Trousseau phenomena are found. Some authors have regarded these cases as cases of latent tetany. The diagnosis of the various epileptiform seizures will be considered in the section devoted to that subject.

The **prognosis** of acute infantile convulsions is generally good, but since death has occurred in these seizures, as well as cerebral hemorrhage, caution should always be exercised in predicting the immediate outcome. The patient having been once tided over the initial paroxysm, it may be confidently expected that it will not be repeated. In the presence of fever, it cannot be predicted what affection may follow the seizure. Primary seizures should not be regarded as forerunners of epilepsy. Many infants and children affected with convulsive seizures pass through later life without any sign of that disease.

Treatment.—The seizure is frequently over before the physician arrives. If such is the case and the infant is in the stage of stupor, it should not be disturbed unless there is high fever or a history of the patient's having eaten some irritating substance. It often happens that the paroxysm supervenes in the presence of the physician. The patient is placed on a bed, the clothes loosened, and a small object, such as the handle of a tooth-brush, placed between the teeth to save the tongue from injury. Nothing further is needed. The paroxysm is as a rule over in three minutes at most. If it persists or is immediately succeeded by another, the patient is placed in a warm bath, after which a few drops of chloroform are administered by inhalation to control the convulsions. A high rectal enema of the temperature of 110° F. (43.3° C.) is at once administered. I have in some cases continued the administration of chloroform for fully an hour. Caution should be exercised in its administration. If, after the seizure, the temperature is high, it is treated as indicated in the section on Infectious Diseases. Unless there is some contraindication, a full dose of calomel is administered as a routine procedure even if an enema has been resorted to. Should the child be restless, it is well after the convulsion to administer a dose of bromide of potassium in combination with chloral, either by mouth or rectum. In repeated convulsions the administration of these drugs during the seizures is of inestimable value.

For several years past I have used the postural treatment in acute convulsive seizures. The patient is placed with the head low, the buttocks raised, and the clothes loosened. I think the paroxysms have been shortened by this treatment. It was suggested by the theory that cerebral anæmia is the cause of the initial paroxysm. I have carried out this postural treatment without any ill after effects, such as hemorrhage. In a large number of cases of repeated convulsions, the postural treatment should be supplemented by chloroform inhalations.

HYSTERIA.

Hysteria is a morbid state of the nervous system in which the primary derangement is in the higher cerebral centres. The lower centres of the brain, the spinal cord, and the sympathetic system may be secondarily disordered (Gowers). It is not a true disorder of childhood. Sixteen per cent. of all the cases of hysteria occur in youth (Steiner).

Etiology.—Hysteria is rarer in children than in adults, is more frequent in the female sex, and is more often seen in boys than in men. According to Briquet and Landouzy, 8 per cent. of all the cases occur in the first decade of life, and 50 per cent. in the second. The cases of the first decade, according to Barlow, generally develop at the age of six years. Cases are occasionally seen in patients of the age of three years. Heredity plays an important etiological rôle. Moral and mental influences predispose to development of the condition. Children of emotional antecedents are apt to be subject to the disease. Sexual disturbances or excesses (as masturbation in boys) are exciting causes. Abnormalities of the sexual organs, phimosis, and hypospadias, are apt to excite masturbation and resultant hysteria. In some subjects, any acute disease, such as pneumonia or typhoid fever, will develop latent tendencies to hysteria. Diphtheritic paralysis may eventuate in hysterical palsy (Gowers).

Symptoms.—The disease shows many variations and most diverse symptoms. The symptoms may be divided into psychic, motor, and sensory manifestations; or into the convulsive and non-convulsive forms of hysteria.

Psychic or Mental Hysteria (Non-convulsive).—In most cases of this class, the patients suffer from some mental strain. The attack begins with a paroxysm of crying or of laughing. The child then passes into a violent condition, striking at persons and tearing the clothes from its body. I saw a case of this kind in a boy eight years of age. He was very bright at school, but shunned the companionship of other boys. He masturbated. At times he was of a very loving disposition, at other times would refuse to do as he was told. The rebellion would terminate in a paroxysm of crying, followed by one of shrieking. The boy would tear his clothes and then calm down quite exhausted. Girls after undergoing some mental strain, such as is incident to a school examination, become irritable, morose, and suffer from insomnia. They have laughing and crying spells and refuse nourishment. After a period of these symptoms they either recover or pass into a state resembling acute mania. Such children are nervous and are born of neurotic parents.

Hystero-epilepsy, catalepsy, or trance symptoms may manifest themselves. These cases are rare in children, but Sachs and Steiner have seen them in children of mentally degenerate families.

Insanity, alcoholism, and chorea in the family predispose to the development of hysteria. These cases must be differentiated from those of true epilepsy.

Motor Manifestations (Convulsive Forms).—These occur in the form of hystero-epileptic attacks. After some mental excitement, a paroxysm beginning with a shriek will supervene, the sounds simulating a bark or a snapping sound. Contortions then supervene and the back is arched, as shown in Richer's drawings. During the attack, which may last for several minutes, there may be no evidence of consciousness. There may be a number of such attacks in the course of twenty-four hours. The patient may suddenly fall down and have contortions, and the attack may terminate in a crying spell. The patients sometimes tear their clothing and become violent. These convulsions are differentiated from true epilepsy in that there is no aura; they are preceded by emotional excitement. The onset is gradual and the patients emit noises of various kinds during the attack. The pupils are normal. There are ecstasy, extravagant movements, and tonic rigidity. The vesical and rectal reflexes are normal. The patients do not bite the tongue, and rarely injure themselves; the loss of consciousness is temporary or imperfect. There are in hysteria irregular twitchings of the extremities and a repetition of one specific movement, such as retraction of the head. The spell or paroxysm ends in a crying or laughing fit, or the patients become melancholic.

Among the manifestations of hysteria in children is the so-called hysterical stricture of the œsophagus, or globus hystericus. There may be spasm of the bladder, hiccough, and loss of voice. The latter is common among young girls. I have seen the children recover their voice under hypnotic suggestion. Hysterical children may, even at the early age of five years, pass under hypnotic suggestion, into a trance-like state. Whether diarrhœa can be caused by hysteria is in my opinion doubtful. I have seen true toxic diarrhœa in neurotic children diagnosed as nervous or hysterical. One case occurred in a boy of six years. Some young girls have attacks in which all varieties of poses are assumed in the nude state. I have seen such a case in a highly intelligent girl of nine years. During the morning bath, the child had a desire to assume the most grotesque poses.

The so-called epidemics of chorea are now known to be simple hysteria. Among these are to be classed the school epidemics and the dancing mania of the Middle Ages.

There may not only be convulsive movements, but also absolute paralysis of single muscles or of a group of muscles. Hysterical paralyses as a rule follow no anatomical distribution. They are distinguished from true palsies by the lack of change in the electrical reactions and in the condition of the deep reflexes. The sphincters are normal. Paralyses, such as those due to neuritis or poliomyelitis, may supervene in a hysterical subject.

The **disturbances of sensation** include hyperæsthesias and anæsthesias. These do not differ essentially from similar conditions in the adult subject. There may be hyperæsthesia in the region of the ovary, or in the skin over the vertebral column. Areas of irritation may cause paroxysms. There are hysterogenic zones which are not hyperæsthetic (Sachs). Anæsthesia, partial or general, is more frequent. There may be absolute anæsthesia to all sensation. There may be blindness in one eye or hemianopsia, deafness, or loss of taste or of smell. Vision may be affected as above described, or there may be photophobia and diminution of visual perception; the retina may be insensible to light, and there may be limitation of the field of vision or temporary bilateral loss of sight.

There are in children cases of anorexia which supervene with vomiting after some nervous strain. I have seen this occur in children who were beginning some course of study. In one case it came on in the morning just before the child started for school. With suspension of school duties, the vomiting ceased. The so-called phantom abdominal tumor seen in rare instances among children may be traced to a hysterical cause. In very young girls I have frequently seen forms of palpitation with cardiac anguish which seemed to be hysterical. Steiner describes these forms of tachycardia. In these cases there is not only absence of cardiac lesion and signs of Basedow's disease, but spinal hyperæsthesia may be elicited.

Diagnosis.—Sensitiveness to pressure over the vertebral column is one of the most frequent stigmata of infantile hysteria (Steiner). Epigastric tenderness is less frequent than among adults. Hyperæsthesia is less marked in childhood than later in life, but is more common than anæsthesia. Jolly says that deep analgesia is rare. Of especial interest in its relation to diagnosis is the fact, that ocular symptoms, such as diplopia, may be present morning and evening. Paralysis may appear and disappear. There are forms in which there may be tachycardia or bradycardia, but during excitement the rhythm of the heart may be normal. Cases have been described in which the headaches, ptosis, and facial palsies simulate the symptoms of tuberculous meningitis. Study alone will clear up such obscure cases.

Duration and Course.—The symptoms of hysteria are not necessarily permanent, but are likely to recur after excitement or nervous strain of any kind.

The **treatment** of hysteria in children is based on the same general principles as in the adult. The child is, if possible, removed from exciting surroundings. Studies are regulated and bad habits, such as masturbation, are, if possible, corrected. The effect of good food and outdoor life is marked. Hydrotherapy and massage achieve their greatest triumph in this affection.

TETANY.

(*Tetanilla; Arthrogryposis.*)

Tetany is an intermittent or persistent, more or less painful, tonic spasm of groups of muscles of the upper and lower extremities.

Forms and Frequency.—John Clark in 1815 described this disease in children. Trousseau, Baginsky, Chvostek, Erb, Escherich, and Ganghofner have completed its symptomatology. In children it is most common from the fourth to the twentieth month. There are two distinct forms. In the first, the contractures are intermittent, and come on at intervals, the patients being free from muscular spasm in the intervals. The second form, now accepted by the majority of writers as the same affection as the former, is that in which the contractures are persistent.

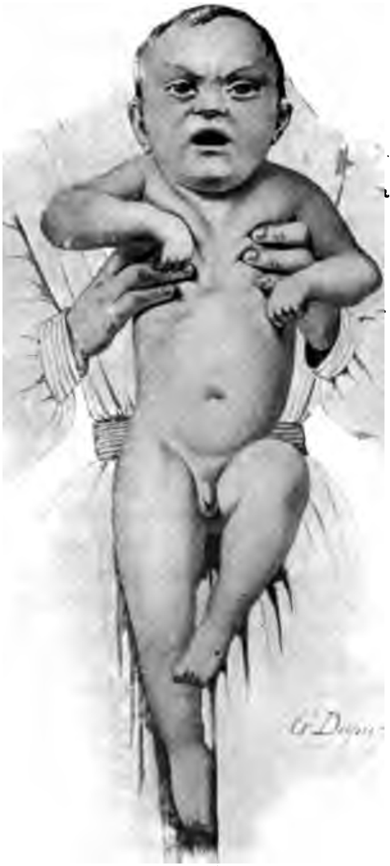
The **etiology** of this affection is still very obscure. It occurs most frequently in the winter and early spring. In my experience in an ambulatory clinic, it was customary to see these cases appear in groups in the early spring months. The affection is seen under the most diverse conditions. Fully 63 per cent. of the cases are rachitic (Fischl). The percentage of rachitis must, of course, vary in different countries, but the cases coming under my notice have been chiefly of that character. The condition is not, as is frequently supposed, a rare one. I have regularly seen a number of these cases yearly. Many cases of tetany are not recognized as such by the physician. Cold, entozoa, infections of the gut, chronic intestinal disturbances of all kinds, rachitis, an enlarged thymus (Escherich), have all in turn been regarded as etiological factors. On the other hand, the general trend of opinion is to attribute the affection to a toxæmia probably originating in the gut and expending itself on the peripheral motor nerves. Fully 73 per cent. of Fischl's cases had shown intestinal disturbances. The fact that the condition occurs in early infancy and in some respects resembles a normal state, to be described later, will account for its being frequently overlooked by the physician.

Morbid Anatomy.—No definite account of the changes in the nervous system or elsewhere has as yet been given. Langhans has described a peri-arteritis and phlebitis in the white commissure and cervical portion of the cord. Gowers, without any positive data, assumes that there are some changes in the motor cells of the cord which cause the increased irritability of the peripheral motor nerves. Fischl in a recent article has published the post-mortem changes in his fatal cases. He makes, however, no comment on them. He found hydrocephalus interna and externa, œdema of the brain and meninges, tuberculosis of the brain, hemorrhagic infiltration of the cerebellum and meninges, chronic intestinal catarrh, and bronchopneumonia. The affection occurs under the most diverse conditions.

The **symptoms** consist of muscular contractures and phenomena connected with the peripheral motor nerves, which are known as Trousseau's phenomenon, Chvostek's facial symptom, and Erb's signs of increased electrical excitability of nerve and muscle.

Muscular Contractures.—These come on without any premonitory symptoms. The infant or child may have been in good health, or may have been suffering from intestinal disturbance. There are two distinct forms of contracture in infants, in one of which the hands and arms take the position assumed in driving horses (Plate XXXI.). The arms are pressed against the thorax, the forearms flexed on the arms, and the fingers tightly flexed over the thumb into the

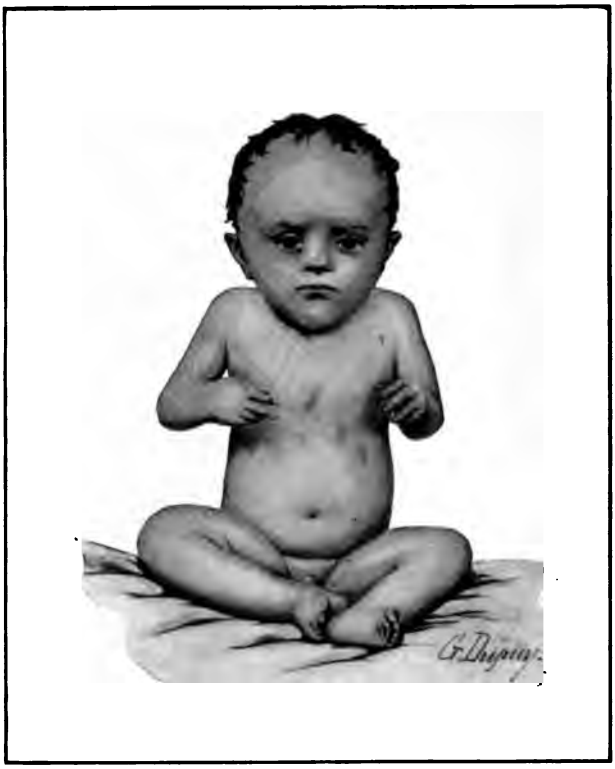
PLATE XXXI.



Tetany. Infant nine months of age. Shows the driving position of the fingers, hands, and arms, overextension of the feet and flexion of the toes.

palm of the hands. The hand itself is strongly flexed on the forearm. The lower extremities may be adducted toward the median line, the thighs flexed on the abdomen, and the legs on the thighs. The feet are as a rule extended in the equinus position and the toes overflexed on the plantar aspect of the foot, the whole foot being slightly curved inward. After the contractures have lasted some time, there is œdema of the tissues over the dorsum of the foot. In the second set of cases the fingers are overextended, as shown in Fig. 167. The arms and lower extremities also take the position of flexion. These contractures are painful; the patient cries as if in great pain when an attempt is made to straighten the fingers or extremities. There may be a temperature of two or three degrees. The contractures may diminish, and there may be an interval in which the only symptoms are such as may be attributed to the increased mechanical and electrical irritability of the peripheral

FIG. 167.



Tetany. Extension of the fingers, flexion of the arms, flexion of the toes. Child, eighteen months of age.

nerves. There may also be eclampsia. The eclamptic attacks are very dangerous. I have lost 2 cases in such seizures. Other muscles, such as the abdominal or thoracic, may be the seat of contracture. In the latter case there may be cyanosis.

I have seen cases in which all the muscles of the body were involved very much as in tetanic conditions. In one case there were stiffness of the muscles of the neck and loss of consciousness. Trismus is rare, and certainly does not occur at the outset, as in tetanus. The muscles of the face may be subject to contracture. The brow is wrinkled, and the face has an anxious expression. If the muscles over the zygoma are tapped, there is an instantaneous contracture or spasm of the orbicularis palpebrarum. In some cases, if the muscles of the face or the forehead are tapped, there is an instantaneous contracture of the muscles of the face, and sometimes of other muscles of the body. This is called the facial phenomenon of Chvostek. If the nerves and arteries at the bend of the elbow are compressed, the characteristic tetany position is produced in the muscles of the hand and fingers. This phenomenon was first noticed by Trousseau, and bears his name. Erb established the fact that there is increased irritability of nerve and muscle to the faradic and galvanic current. If the muscles or nerves elsewhere in the body are tapped, or if pressure is brought to bear at the point of exit of the nerve-trunks, there is an excessive irritability to this mechanical stimulus. The knee reflex is increased.

Duration.—The disease may last a few hours, days, or weeks. In many cases the contractures disappear for a time, leaving the patient perfectly free from symptoms. They may return in all their original severity. The attacks leave the peripheral nerves in a condition of increased excitability. In such cases both the Chvostek and Trousseau phenomena may be present.

The **diagnosis** of fully developed tetany is based on the presence of muscular contractures, of increased mechanical irritability of the peripheral nerves (as evinced in Chvostek's symptom), and the presence of Trousseau's phenomenon. There are cases of tetany in which the facial symptoms are lacking. On the other hand, I have, in cases in which there was laryngospasm without contractures, obtained both the facial and Trousseau phenomena.

The Relationship of Laryngospasm to Tetany.—Escherich, his pupil Loos, and also Ganghofner, have recently called attention to the fact that laryngospasm is present in a certain number of cases of tetany. They also found that cases of laryngospasm which did not present contractures, did show the facial phenomenon of Chvostek and the Trousseau symptom. They concluded that laryngospasm was a manifestation of tetany, whether the muscle contractures were present in the extremities or not. Their observations have been amply confirmed, but not all observers are as yet willing to accept laryngospasm without contractures of the muscles of the extremities as true tetany. The views of Kassowitz and Hochsinger are at variance with those of Escherich. They consider rachitis the fundamental cause of laryngospasm, if not of tetany.

The term *latent tetany* has been applied to those cases which show no muscular contractures or laryngospasm, but in which the facial Trousseau or Erb phenonemon may be elicited, or in which the mechanical, and especially the electrical, contractibility of muscle and nerve are increased.

Prognosis and Mortality.—The prognosis in the sporadic cases is very good. Parents should be cautioned in regard to the excitability of the patient and the possibility of eclampsia, with its fatal consequences. I have lost 2 cases in convulsions. The persistent cases may be complicated with other affections, such as tuberculous meningitis. If such is the case, the outcome is, as in the primary disease, fatal. Epidemics in hospitals for children present unfavorable features; Escherich lost 37 per cent. of his cases.

Treatment.—The bowels should first be evacuated. Calomel is given in grain ½ (0.03) doses two or three times daily. If there is any disturbance of the gut, the patient is given a high enema once a day. Milk is suspended until the movements take on a more favorable appearance. The infant is kept under the influence of the mixed bromides of potassium, sodium, and ammonia. If there is eclampsia or increased irritability, a warm bath is given at least once a day. The patient is kept quiet and not disturbed much. Nc attempt to straighten the limbs should be made, since it causes pain.

CATALEPSY.

Epstein has recently described a condition in children closely resembling a similar affection in the adult. He has described it as catalepsy occurring in infants poorly nourished and rachitic. The ages of his cases ranged from eighteen months to three and one-half years. Epstein believes there is a disturbance of the psychomotor functions. The phenomenon was observed by him chiefly in the lower extremities. Either extremity on being lifted into the air would stay there for a length of time in any position of flexion or extension in which it was placed. This phenomenon was not present during sleep, nor was it accompanied by any muscular rigidity or increase of mechanical or electrical irritability of the peripheral nerves. I have met a marked case of catalepsy following an attack of typhoid fever in a boy of four years. The hands, arms, and lower extremities would remain for long periods of time in the position in which they were placed. The boy would sit for long periods staring ahead, without winking the eyes.

MYOTONIA.

Myotonia physiologica neonatorum is a term applied by Hochsinger to the normal tendency of the newly born infant to flex the fingers, arms, and lower extremities. There is a slight rigidity which is a hypertonicity of the muscle, and which lasts until the third month. The position closely resembles that of the extremities of the fœtus in utero. The myotonia is exaggerated if the infant becomes ill with any intercurrent affection, such as syphilis. The condition cannot be mistaken for tetany if the differences between the normal and the abnormal states of the peripheral nerves are borne in mind.

CONGENITAL STRIDOR OF INFANTS.

(THOMSON.)

This rare condition has for a long time been classified by writers as a mild form of laryngismus stridulus. I have seen one case in which there was also laryngismus. The affection is a distinct one, is generally congenital, and appears soon after birth. Some years ago, I presented a case of the kind before the Pediatric Section of the Academy of Medicine of New York. Since then I have seen a number of cases. Thomson has fully described and studied the affection. The infant is usually in other respects normal, but I have seen the condition in infants with signs of rachitis. The ages of the patients varied from nine weeks to twelve months. In one case there was a history of attacks of laryngismus stridulus, occurring shortly after birth. In most of the cases, the symptoms were noticed soon after birth. The respiration is more or less noisy, being sometimes scarcely audible and at other times so loud as to be heard at some distance. Inspiration is accompanied by a peculiar croaking, grunting noise. As a rule, expiration is noiseless, but it may be accompanied by a grunting sound, there being short intervals in which no sound is heard. The infants are not at all disturbed by the condition. They sit and play, emitting this peculiar croak while breathing. In mild cases, nothing is seen in the thorax. I have, however, seen the drawing inward of the suprasternal region which Thomson describes. In one case the noise was louder at night. If the stethoscope is held over the situation of the vocal cords, it will be ascertained that the sound is produced in the larynx and not in the pharynx.

The causation is obscure; the theory advanced by Thomson is that there is an ill-coördinated spasmodic action of the muscles of respiration, choreiform in character and similar to that present in stammering. This influence acting on the epiglottis from birth causes a deformity of the organ, which in turn perpetuates the crowing noise. Others have attributed this condition to the presence of an enlarged thymus (Variot). Some of these infants are distinctly lymphatic, and Hochsingen has lately with *x*-ray demonstrated what he believes to be an enlarged thymus in many of the cases of laryngeal stridor coming under his notice. He believes the condition due to an enlarged thymus, and suggests that the term "Asthma thymicum" be applied to these cases. Lee and Refslund have published two cases with autopsy in which laryngeal stridor existed from birth and in which there was an anatomical malformation of the epiglottis. This consisted in a folding of the epiglottis laterally, so that the aryepiglottic folds were almost in contact. The superior opening of the larynx was thus covered by the deformed epiglottis in such a way that respiration took place through a mere slit of epiglottis, hence the grunting or sawing noise. I have recently published a case of laryngeal stridor dying of intercurrent pneumonia. This case showed the same malformation of the epiglottis described by Lee and Refslund, and would support the theory of anatomical deformity as a causative factor in these cases. Toward the second year of life the condition gradually disappears spontaneously.

LARYNGISMUS STRIDULUS.

(*Spasm of the Glottis.*)

Laryngismus stridulus is a spasmodic functional nervous disorder of the glottis, involving the muscles of inspiration and expiration.

Occurrence.—The affection is more frequent in boys than in girls. It is most common in the first year of life. The majority of the cases occur before the end of the second year. Kassowitz found 348 of 370 cases to occur before that time. It may occur in the newly born infant (Henoch, Kassowitz). Most of the infants and children affected by this disorder are subjects of rachitis and also show signs of craniotabes. Henoch estimates the frequency of rachitis at 75 per cent. Only one of the cases of Kassowitz did not show its signs. All but 48 showed craniotabes. On the other hand, Boral shows that 4 per cent. of all children with rachitis have laryngismus stridulus.

The **etiology** of this affection is obscure. Although rachitis is so frequent an accompaniment of the disorder, it may not yet be assumed that it is the exciting cause. Craniotabes, which is a part of the symptom-complex, has been regarded as the cause (Elsässer).

Escherich, Loos, Gee, and Ganghofner have placed laryngismus stridulus in the same category as tetany, and trace it to the same exciting cause. Reflex irritation from the stomach acting through the vagus, is the theory of Baginsky. In many cases which have terminated fatally an enlarged thymus has been found. On the other hand, there have been post mortems which showed a rather small thymus and slightly enlarged broncial nodes (Baginsky).

Morbid Anatomy.—No definite study has been made of the changes found in the fatal cases. Most cases show œdema of the brain and some fluid in the ventricles, rachitis slight or pronounced, the thymus small or enlarged, and the lymph-nodes slightly enlarged. The cases with enlarged thymus thus far published have not been convincing. Children with enlarged thymus die of other disorders, and without having had during life any symptoms of spasm of the glottis.

Symptomatology.—The spasm or paroxysm comes on suddenly. Without the least warning, the child throws the head back and stops breathing; the face becomes livid, the arms are flexed and the hands clenched. No respiratory movement takes place for a few seconds. There is then a long-drawn whistling or crowing inspiratory sound. This is the classical form of spasm of the larynx. The paroxysm may begin with a piping, inspiratory sound. Apnœa lasting for a varying length of time succeeds, and is followed by a loud or silent expiration. Apnœa may appear first, and be followed by several noisy explosive expiratory movements, which may be succeeded by several noisy crowing inspiratory sounds. The picture is usually that of spasm of the glottis as first described, in which the breathing stops entirely. The attack may come on during absolute quiet or during sleep, the onset of the attack causing the child to wake. The paroxysms may be brought on by excitement, a draught of air, or by pressure on the larynx. They are of all degrees of severity. Some infants show a form which is very disquieting. In a fit of crying the child takes a number of noisy inspirations and expirations, and then stops breathing, becomes cyanosed, clenches the hands, and threatens to pass into an eclamptic paroxysm (expiratory apnœa), when suddenly a deep inspiration occurs and the danger is passed. Some cases of the classical form have eclamptic seizures. There may be convulsions, especially in the form described as expiratory apnœa. One of my cases was that of an infant a year old, one of twins. The infant was anæmic, and showed marked signs of rachitis and craniotabes. It was in apparent health until the eighth month of infancy, when attacks of respiratory apnœa appeared at first at intervals of three weeks, and finally daily. The infant during a crying-spell would stop breathing, become cyanosed, the left hand and arm and lower extremity and muscles of the face contracted in tonic spasm, during which the heart became very slow in action and irregular. The left-sided spasm lasted for a few seconds, and then the infant relaxed and quietly passed into a sleep, from which it awoke in a few moments. In all of these cases there is the ever-present danger that the glottis and the muscles of respiration, including the diaphragm, will fail to relax, thus causing death with convulsions. The number of attacks of spasms of the glottis may reach twenty or thirty a day, or they may be very infrequent, occurring only once every few days, weeks, or months. In all the forms, including the classical one just detailed, the spasm involves not only the glottis, but also the diaphragm and other muscles of respiration. The infants may show no symptoms after the paroxyms. On the other hand, some infants seem to be overcome and pass into a stupid state lasting for fully ten minutes. It is difficult to estimate the degree of consciousness during an attack, but even in the mildest forms there may be a momentary loss of consciousness (Henoch). Most cases show the facial and Trousseau symptoms of tetany and increased irritability of the peripheral nerves.

The **prognosis** of spasm of the glottis is good. The danger lies in the eclampsia, during which death may supervene.

The **diagnosis** is not difficult. There are all degrees of severity of the spasm, ranging from partial to complete closure of the glottis. In the latter form a rachitic infant in a paroxysm of crying is frequently heard to give several inspiratory crowing sounds without having any further symptoms. There is a species of laryngeal incoordination. These cases may at intervals develop typical paroxysms. The parents should be warned of this possibility. The forms of spasm of the glottis which have just been described should not be confused with spasm or difficult breathing due to pressure of a retropharyngeal abscess or suppurating gland upon the larynx.

Complications.—Pertussis may complicate a case of spasm of the glottis. Cases thus complicated give a grave prognosis (Henoch). Tetany has been elsewhere mentioned as an accompanying condition.

Treatment.—During the Attack.—The infant is carried to an open window. A draught of air is allowed to blow in its face or a few drops of water are thrown in the face. This is done to excite a reflex relaxation of the glottis. The head should be held low, as in ordinary eclampsia. If relaxation of the glottis does not occur and convulsions set in, a few drops of chloroform may cause the muscles of respiration and those of the glottis to relax. Intubation and tracheotomy have been performed at this crisis, when the breathing threatened to cease permanently. If, however, as sometimes happens, the muscles of respiration are also involved, the paroxysm will occur with the tracheotomy tube in the trachea. Störk has published a case in which the insertion of a tracheotomy tube had not the least influence on the paroxysms. This is a very important observation, and raises the question of the propriety of intubating or performing tracheotomy. On the other hand, cases have been intubated and resuscitated with artificial respiration (Pott). In the intervals, the treatment should be chiefly directed toward the rachitis. The feeding should be carefully attended to; the infants should, if possible, be breast-fed. Bottle-fed infants should be fed on raw milk, beef-juice, orange-juice, cereals, and eggs. The medicinal treatment which in my hands has given the best results has been the administration of an albuminate, or peptonate of iron or manganese in full doses. To prevent the recurrence of the laryngismus or apnœic attacks, full doses of the mixed bromides are given. To an infant one year of age as much as 5 grains of the mixed bromides of sodium, potassium, and ammonium are given three times daily, and continued over some period of time. Under this medicinal treatment I have been able to control apnœic attacks. In my hands the administration of phosphorus has not been attended with any success.

Bathing in cold water has not in my experience been productive of good results.

EPILEPSY.

Epilepsy is not a disease peculiar to infancy and childhood. It is discussed here simply to emphasize the peculiarities of the affection as seen in children. It is a true disease of the nervous system, and has nothing in common with and no demonstrable relationship to infantile convulsions. Fifteen per cent. of the cases of epilepsy occur before the fifth year of life. Henoch has seen a case in an infant one year of age who had convulsions beginning with a cry and during which the infant bit the tongue. He describes another case in a child three years of age, in which the attack began with vertigo. In another case, in a child three years of age, the patient fixed a point and ran blindly toward it. The latter appears to have been a case of "procursive epilepsy."

Etiology.—According to Gowers, in two-thirds of the cases of epilepsy in children the parents are neurotic and hysterical. Chorea in the mother will often manifest itself in epilepsy in the child. Infantile palsy or traumatism is more frequently than heredity the cause of epilepsy. Epilepsy following slight palsy is likely to be mistaken for hereditary epilepsy.

Symptoms.—In children, as in the adult, there are no symptoms in the intervals between the attacks. Only such results of attacks as a bitten tongue or local traumatism are seen. There are, as in the adult, two distinct forms of epilepsy—grand and petit mal—between which there may be all variations participating in the peculiarities of both forms. In grand mal there is the aura, sensory or psychic; it is present in a large percentage of the cases in children.

Baginsky calls attention to a case in which epigastric pain was the aura preceding the attack. The other forms of aura are numbness and tingling of the extremities, general restlessness and irritability and auditory phenomena in which a peculiar cry of an animal is perceived. There may be a hissing sound. An aura referred to the sense of taste is very rare, and most neurologists do not make note of having found it in any case. In children the perception of peculiar odors just prior to the attack occurs as a form of aura.

After the aura, the attack begins with a cry followed by sudden loss of consciousness and tonic or clonic spasm of the muscles, which may be unilateral, general, or partial. The pupils dilate; there is spasm of the respiratory muscles and those of the jaw, as well as foaming at the mouth and biting of the tongue. The spasm then relaxes, the movements become first clonic and then intermittent, there is involuntary passage of urine and feces, and consciousness gradually returns, the patient passing into prolonged stupor and profound sleep. Some of these symptoms may be absent, but the loss of consciousness, dilated pupils, spasm, and the succeeding profound sleep are constant. In the majority of cases, the presence of any two of these will be sufficient for making a diagnosis.

Convulsions.—General convulsions indicate hereditary epilepsy. Convulsions may at first be partial, but in the majority of cases eventually become general. Partial convulsions indicate disease in the motor areas. The attacks taking the form of petit mal may be so slight as to be mistaken for fainting spells. Such attacks may occur in young children. One of my cases was in a child five years of age. An epileptic spell is momentary; a fainting spell is gradual, there are no vasomotor disturbances, and the pupils do not dilate. Henoch and others record cases in which the children momentarily stop the occupation in hand, stare into vacancy, and then recover themselves without having any recollection of the interruption. In other cases there is an irritable attack or mild maniacal outbreak. In .some cases the child passes into a state of mental confusion in which it performs acts unconsciously. Attacks of double consciousness or narcolepsis are rare in children (Sachs).

Attacks of grand mal are sometimes associated with a rise of temperature. A case recently came under my observation in which a girl of eight had as many as forty convulsive seizures in twenty-four hours. There was a slight rise of temperature which could not be traced to any cause other than the convulsions. Thomson and Oppenheim have shown that there are a concentric limitation of vision and a diminution of general sensibility for some time after the epileptic attacks.

Diagnosis.—Epilepsy must be differentiated from syncope, hysteria, post-hemiplegic convulsions, and tumor of the cerebrum. The peculiarities of an attack of syncope and hysteria have been dilated upon. The post-hemiplegic convulsions will, in the intervals, reveal the paralyses and contractures with increase of deep reflexes. Attacks of convulsions caused by tumor are confined to groups of muscles if the tumor is in the motor area, and are combined with optic neuritis if the chiasm is directly or indirectly the seat of pressure.

With tumor, there are in the intervals peculiarities of the gait which aid in diagnosis.

The **treatment** of epilepsy is essentially the same in children as in the adult subject.

PAVOR NOCTURNUS.

(*Night-terrors.*)

There are two forms of this affection—the primary or idiopathic and the symptomatic form. In both, the children retire to sleep and after an hour or two suddenly awaken from deep slumber with a shriek or cry. They are pale, greatly terrified, and grasp at the empty air. In incoherent, broken phrases they try to collect their thoughts. Some children see terrifying visions and either cling to the bystander for protection or try to get out of bed to escape an imaginary danger. After being quieted the children fall asleep, and when questioned the next morning have no distinct recollection of what has occurred. These attacks may occur every night for days, weeks, or months. They rarely occur twice in the course of the same night.

The idiopathic form of this affection may occur in children who are naturally of a nervous temperament without any apparent exciting cause. I have seen it in children who were distinctly the opposite of nervous, and who were well nourished and good natured. The night-terrors may follow epilepsy or they may be so severe as to be the exciting element in precipitating an attack of chorea. Children sometimes have real hallucinations, which may be present even during the day (Henoch). It may, however, be said that hallucinations during the day are really not included in the idiopathic form. This affection occurs chiefly up to the time of second dentition. Forms of terror in older children are hysterical. Adenoids are supposed to be an etiological factor, but this is doubtful. It is only in the symptomatic form that children, after having committed some error in diet, awake with the symptoms above described.

The **prognosis** is good. The affection never precedes insanity. It subsides under treatment or disappears spontaneously.

Treatment.—In the symptomatic form, the meals should be so arranged that the lightest repast is that taken in the evening. In the idiopathic form, bromide of potassium is most useful. It is administered in one dose, an hour before retiring. The children should not be too active mentally during the daytime. Visitors should be restricted to certain hours. Play and sport in the open air are indicated. The school tasks of older children should be completed in the afternoon.

CHOREA.

(*St. Vitus' Dance; Sydenham's Chorea.*)

Chorea is a nervous disease characterized by irregular involuntary movements or twitchings of some or all of the muscles of the body. It is accompanied by muscular weakness and mental disturbances. In some cases there is endocarditis.

Classification.—Chorea minor is an acute disease described by Sydenham. Chorea major is a hysterical disorder; under this heading are included the chorea electrica, and the dancing mania with rhythmical motions, of the Middle Ages.

Chorea insaniens is the fatal form of acute chorea minor.

Laryngeal chorea is a hysterical affection (Gowers).

Choreiform affections or pseudochoreas comprise the cases of tic convulsif of French writers and other forms of habit-spasm, local or general.

In addition there are forms of chorea which are symptomatic or secondary to infantile palsies. Huntington's chorea is a chronic progressive affection of a hereditary nature.

All these forms of chorea except chorea minor and insaniens should be excluded from the category of Sydenham's chorea.

The epidemics of so-called chorea, occurring in schools, are probbly hysterical disorders which are the result of imitation and not true Sydenham's chorea.

Frequency and Etiology.—Chorea is more common among female than male children. Of 554 cases collected by Osler, 70 per cent. were of the female sex. It rarely occurs before the fourth year. Starr's statistics of 1400 cases show 8 at the third year. Cases are recorded as occurring in newly born infants, but are not accepted by all authors as authentic. The disease is most common from the fifth to the fifteenth year. Fifty per cent. of Starr's 1400 cases occurred before the tenth year, and 75 per cent. from the fifth to the fifteenth year. Of 83 cases of chorea occurring in my ambulatory and hospital service, 23 were of the male and 60 of the female sex. Ten children were under the age of five years, and 67 cases occurred from the fifth to the tenth year. Thus, the greatest frequency is at the latter period. Only one case occurred in a very young child (two and one-half years). The disease is found in children in all walks of life. Children of a nervous, ambitious temperament with a hereditary neurotic history are more prone to contract this disorder than those of a more equable disposition. It is therefore more common in towns and large cities than in country districts. In some cases there is a history of fright or traumatism, either immediately preceding an attack or coincident with its onset. It is as yet impossible to say, however, whether there is any relation between chorea and these occurrences. They may have some influence in developing latent tendencies to the disease. An attack will often be initiated by a scolding or chastisement on the part of parents. The spring months show the greatest number of cases, the least number occurring in the late autumn. There also appears to be a correspondence in the prevalence of cases of chorea and rheumatism at certain periods of the year (Osler, Lewis). The relation of a condition of lymphatism (adenoids or nasal catarrh (Jacobi)) to true Sydenham's chorea is not generally accepted. Errors of refraction in the eyes also seem to be a predisposing cause of the outbreak of choreic attacks. (de Schweinitz). These can scarcely be regarded as a direct cause of Sydenham's chorea, but acute articular rheumatism may be so considered. Rheumatism seems to run in families in which the children have chorea. Osler finds that 15 per cent. of his cases are of such families. Of the subjects of chorea, fully 21 per cent. show a history of rheumatism (Osler). These figures correspond more or less to the statistics of Townsend, 21 per cent.; Starr, 21 per cent. in 1400 cases; and my own cases, 18 per cent. Crandall gives the highest frequency of rheumatism in cases of chorea (54 per cent.). In the majority of cases the rheumatism precedes the chorea (Sée). I have seen one case of chorea preceding an attack of rheumatism in a child four years old. I believe that, with cases of rheumatism of the acute articular type, there should also be included those of articular pains without swelling of the joint. The forms of rheumatism with chorea giving the so-called subcutaneous fibrous rheumatic nodules are rare in this country (Osler).

Chorea may complicate any acute infectious disease, such as scarlet fever, whooping-cough, measles, diphtheria, typhoid fever, and forms of sepsis. There are, however, no definite data of the exact relation, if there be such, between chorea and the infectious diseases. The theory that an attack of any of these diseases will cut short an attack of chorea is not borne out by clinical experience (Henoch).

Morbid Anatomy.—The pathology of chorea is still incomplete and can therefore be merely indicated. Hyperæmia of the brain and cord were found by Pye-Smith and Ogle. Anæmia and proliferation of connective tissue were recorded by Steiner. In the cases of Meynert there was hyaline degeneration of the nerve cells of the central ganglia. Flechsig mentions hyaline degeneration of the lenticular nucleus. Dana studied some cases in which he found hyperæmia of the brain, and degenerative changes in the walls of the bloodvessels of the white substance, with perivascular exudation and accumulation of leucocytes. Jackson has advocated the embolic theory (endocardial). At present there is a great preponderance of evidence in favor of the infectious theory. Berkeley found staphylococci in the blood in a fatal case of chorea. In another case, Naunyn found cladothrix in the meninges and endocardial vegetations. It is certain that just as rheumatism and endocarditis are infectious diseases, so chorea in many cases can only be understood on that theory. Cesaris-Demel has experimentally shown that the central nervous system is peculiarly susceptible to certain pathogenic micro-organisms and their toxins. The staphylococcus and its toxins when injected experimentally under the dura mater cause the formation of small foci of inflammation, and symptoms very similar to those of chorea.

Symptoms.—Children will at the outset of this disorder exhibit mild symptoms of nervous irritability, will be cross, have outbreaks of peevishness and temper, will drop things, and be generally careless in their habits. There is sometimes a history of night-terrors or morose crying spells. There is likely to be loss of appetite; headache is not uncommon, and there may be pains in the limbs or joints and general restlessness. The disease may begin in a certain set of muscles, or in the muscles of one-half the body and thence spread to the whole trunk. Of 301 cases of the statistics of Sachs, there was hemichorea or involvement of one set of muscles in 67. Of Starr's 1400 cases, 951 were general and 449 unilateral, the right side being affected more frequently than the left. When fully developed, the picture presented by these patients is so characteristic as to be easily recognized. On the other hand, the popular notion, so prevalent even among physicians, that every twitching is choreic, has led to grave errors. The following are the main symptoms:

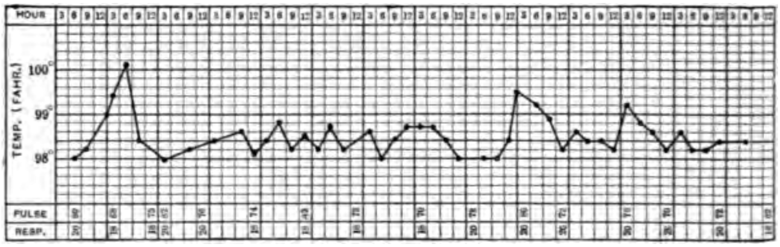
Motor.—The twitchings usually begin in the right hand, only rarely in the legs. After a time there are incessant, irregular, awkward twitchings of all the muscles of the body, which are intensified by volition. If the child is directed to stand still, with the feet together and the arms and hands held out at right angles to the body, the motions are intensified. If it is told to close the eyes, there is a distinct swaying of the body. The movements are not only irregular, but awkward. The patients trip in walking, upset their food and drink, and cannot button their clothing with ease. As a rule, the muscular twitching ceases in sleep, but it may persist. The muscular power is weakened, although distinct paralysis does not occur. The muscle is more paretic than paralytic. Some children let the arm hang at the side. There is wrist-drop when the children are asked to hold out the arms. The tongue is affected in all cases. Sachs places much diagnostic value on the choreic movements of that organ. When children are asked to show the tongue, they will protrude the organ with a jerk, then withdraw it and twist it here and there in the cavity of the mouth. When the tongue is held out quietly, fibrillary twitchings in the organ may be detected. Electrical reaction or irritability of the muscles in chorea can be tested only when the disease is unilateral. In some cases there is no change. In others, according to Gowers, there is a distinct increase in the galvanic and faradic irritability of nerve and muscle. The muscles of the hands, face, and extremities are all involved in the twitchings of the voluntary muscles. The involuntary muscles, such as the cardiac muscle, are not affected. Their involvement has long been a matter of discussion.

Disturbances of sensation are not common. Children have the arthritic pains. Numbness, tingling, pricking, and anæsthesia of the pharynx are recorded. Attacks of multiple neuritis and epileptic seizures should be regarded as complications. The reflexes are not markedly affected. They may in rare cases be slightly diminished or increased (Henoch). Any marked change in the reflexes may be traced to changes of an organic nature, in the cord. The occurrence of headaches or eye-strain as concomitant conditions has been referred to.

Urine.—The urine may contain albumin. Cases with nephritis as a complication have been reported (Thomas).

The **speech** is affected in 25 per cent. of the cases. The patients hesitate and mumble their words or there is difficulty of phonation due to inco-ordinate action of the larynx. Laryngeal chorea, in which there is a distinct sound resembling a bark, is seen in rare cases. It is classified by Gower as a hysterical disorder, truly choreic. I have never met a case of the kind in a child. Deglutition may be affected because of the muscular inco-ordination.

FIG. 168.

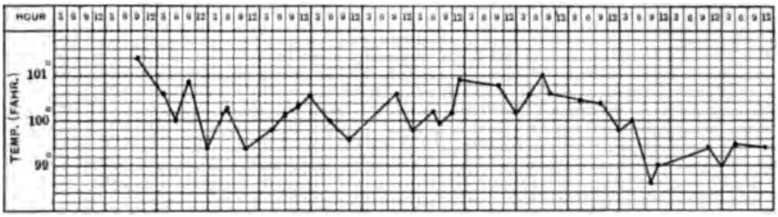


Chorea. Recurrent attack of moderate severity. Systolic murmur over the aortic area. Fourteen days of the temperature is shown here. Child, twelve years of age.

The **cardiac symptoms** are the most important clinical feature of chorea. There is very little doubt, that in a fixed proportion of cases, rheumatism plays an important rôle and that the rheumatic poison, whatever it may be, expends its force upon the endocardium and pericardium. In 20 per cent. of the cases of Osler and in 12 per cent. of Starr's material, organic lesions of the heart were found.

The frequency of cardiac disease in chorea varies as given in hospital and ambulatory statistics. The severer cases come to the hospitals. The majority of the ambulatory cases are mild. Thus 39 per cent. of my hospital cases showed a cardiac lesion (endocarditis), while only 13 per cent. of the ambulatory cases were similarly affected. There would thus be an average of 26 per cent. of both hospital and ambulatory cases. The lesions in simple chorea referable to the endocardium usually affect the mitral valve. Of 17 valvular lesions, 14 occurred at the mitral valve (systolic). The aortic valve was affected in 3 cases (Fig. 168). Pericarditis occurred in one of my cases. In the majority of cases in which there was endocarditis either the patient or the parents gave a rheumatic history. On the other hand, not all murmurs of the heart are organic. In 9 per cent. of Starr's 1400 cases, there were functional murmurs heard at the base and over the pulmonic area, early or late in the disease. A gentle blowing at the apex which is heard to the left of the sternum and is not conducted into the axilla or arteries is heard late in the affection, and is undoubtedly hæmic or myocarditic (Osler). I have heard these murmurs in many cases and have come to the same conclusion. Murmurs may also arise at the tricuspid orifice. The organic murmurs are, as stated above, produced at the mitral orifice in the greatest number of cases. They may arise in the course of the disease or may appear during a relapse. Such cases will show a temperature (Fig. 169). The temperature may after a time become normal, and, in a week or more, while the chorea is still in progress there may be a rise lasting for a day or more, after which it may then again subside to the normal. The temperature may be but a fraction of a degree above the normal, and the diurnal course may be distorted or subnormal (Jürgensen). There is thus clinically a true endocarditis. This form of endocarditis may pave the way for future chronic valvular disease. Under the heading of Chorea Insaniens, I have noted two fatal cases of this form of heart disease. Chorea

FIG. 169.



Chorea. Endocarditis. Previous attack six months prior to the present illness, which was of five weeks' duration before the above observation. Pains in the joints, especially the knee. This curve shows two weeks of the endocarditis. Recovery. Female child, five years of age.

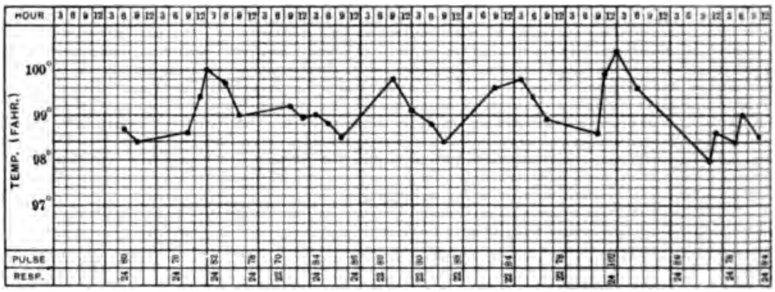
of the heart muscle is not clinically recognized. Pericarditis with endocarditis may occur in cases of recurrent chorea. I have seen two such cases. Functional disturbances such as palpitation and arhythmia also occur.

Temperature.—There are some forms of chorea minor without any signs of endocarditis, which run a course with a slight temperature, the cause of which is undetermined. Some authors think that there may be a latent endocarditis in these forms of chorea (Henoch). If endocarditis is present, there may be a temperature only slightly above normal. In most cases of chorea, there is no temperature (Fig. 170). Fatal cases of chorea, with few exceptions, show signs of endocarditis. Osler has made a study of 80 such cases, and found only 5 which post mortem did not show changes in the valves.

The mental symptoms are in some cases marked. The patients show apathy and depression. The children often, while they are under treatment, have spells of mental depression and fits of crying. It is only in the cases of insaniens, that delirium occurs. In severe cases, there is a period of more or less mental depression, extending far into convalescence.

The **diagnosis** of chorea minor is not difficult in the majority of cases. The picture is a very characteristic one. There are slight twitchings, which so closely resemble habit movements that it is often not easy to come to a conclusion in regard to them. Sachs thinks that the twitchings of the tongue are a means of distinguishing the mild cases of chorea from cases of habit movements. If the patient is told to show the tongue, the tremors and twitchings of that organ and the facial grimaces at once become marked. The movements of the muscles are more rhythmic in hysteria than in chorea. True Sydenham's chorea should be distinguished from the chorea and athetoid movements seen in cases of infantile palsy. The history of the cases, the paralysis, the condition of the reflexes and

FIG. 170.



Chorea, without endocarditis, two months in duration. No rheumatic history. Female child, nine years of age.

the contractures will be of assistance in making a diagnosis. True Sydenham's chorea should also be differentiated from cases of tic convulsif and habit movements. A diagnosis of chorea, made in a case which has lasted for a year or more, is open to doubt.

The **duration** of chorea is variable. It may last from three to ten weeks, and may recur. The recurrent attacks are not necessarily any more severe than previous attacks. Fully one-third of the cases in some statistics show two or more attacks. Of Starr's 1400 cases, 365, or 26 per cent., had relapses. One case had nine attacks. Starr thinks relapses less frequent in private practice than in hospitals.

The **prognosis** of chorea minor is very good. Recovery is the rule, but in exceptional cases it may be delayed for fully three months.

The **treatment** of chorea consists at first in giving the patient perfect rest and quiet surroundings. Children are put to bed and kept free from excitement. I do not think it necessary to isolate them, and it is not wise to do so, since they may, under such treatment, become melancholic. An ordinary amount of quiet, such as is prescribed in cardiac cases, is all that is usually necessary. The patient may be allowed to look at picture-books, but not to study or to read. A simple, easily assimilable diet is indicated, milk and eggs being the chief articles. A warm bath is given daily and the spine sponged with cool water, as some authors recommend. I have not found this necessary in all cases, and would advise it to be omitted if the children strongly object to it. Massage is of great value with anæmic children in whom the circulation is below the average and who have no cardiac disease and no temperature.

Drugs.—Fowler's solution is used almost as a routine remedy in these cases. In my experience its curative effects are doubtful. I therefore prefer to give it in small tonic doses, rather than risk the ill effects of large dosage. There are cases in which any attempt to administer it causes vomiting, and which therefore do much better without it. In any case it should be well diluted. In this way larger doses can be given for a greater length of time than would otherwise be possible.

Cases which show recent or old endocarditis or which have articular pains should receive antirheumatic treatment. Alkalies to keep the bowels open, alkaline baths, and sodium salicylate are the remedies in use in these cases.

If there is great restlessness, bromides should be resorted to. It is a very good plan to combine the bromides of sodium, potassium, and ammonium in one mixture. Trional given in grain v (0.3) doses several times daily is a very good remedy in this set of cases, especially if there is wakefulness at night.

If on account of the loss of appetite and general mental depression it is not possible to give any drugs, the children are simply kept quiet and given a nutritious diet. They frequently recover without the help of any drugs. In ordinary cases there is no necessity of using opiates, such as codeine. Antipyrin in grain v. (0.3) doses has been recommended. I have not found it better than other remedies. Children who have recovered should be kept quiet for fear of a recurrence of symptoms. This is especially true of cases in which the heart has been the seat of a recent endocarditis.

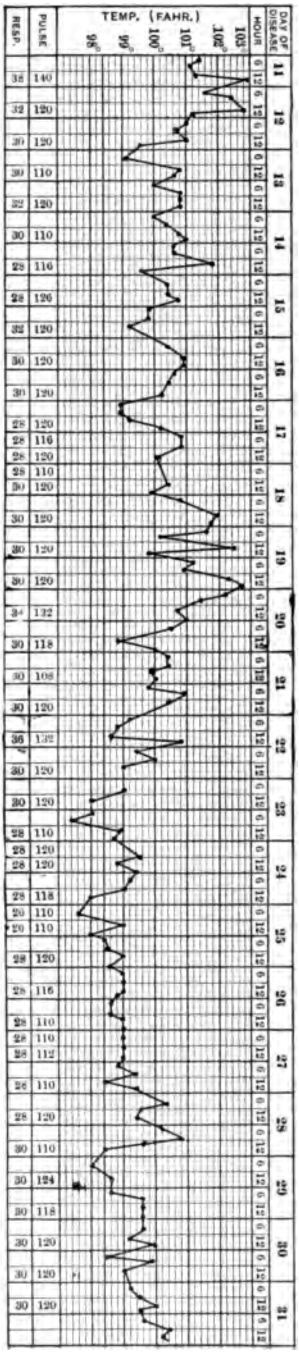


Fig. 171.

Chorea insaniens. Fourth attack of chorea, pericarditis, endocarditis. Recovery with a weakened mental condition. Boy, six years of age.

Chorea Insaniens.

Chorea insaniens is a term applied to the severest form of chorea. A large number of these cases run their course with delirium and high fever. It occurs especially in female subjects. At the outset there may appear to be nothing more than an ordinarily severe chorea, but the patient rapidly becomes worse. Delirium with hallucinations sets in, finally giving way to incoherency and mania. The patients are in incessant motion and do not sleep at night. The fever may mount as high as 107° F. (41.6° C.). The cases are in many instances fatal. Osler gives a *résumé* of some fatal cases. I have seen 2 fatal cases of this form. One case occurring during my service as interne at Bellevue Hospital was that of a girl of twelve, who died with symptoms very similar to those of acute mania. Another case, seen recently, was a boy of ten years, who had for two years previously suffered from ordinary chorea. He had a mitral regurgitant murmur. Two weeks before his death he was suffering from a mild recurrence of the chorea. While in that state he was operated on for adenoids and enlarged tonsils. Chloroform was administered. Three days after the operation the boy was taken with a chill the chorea became worse, and there was fever. Examination of the heart showed endocarditis and pericarditis with dilatation of the left ventricle. In the second week the boy became delirious and did not sleep at night. He complained constantly of pain in the præcordium and tossed in bed. He died two weeks after the onset of the disease. There was throughout a high febrile movement. A third case was that of a boy six years of age, whose temperature-curve is herewith appended (Fig. 171). This case occurred in my hospital service. It was the boy's third attack of chorea. He had chronic cardiac disease. In the final attack there was complicating pericarditis with effusion. The delirium was constant and the choreic movements incessant. He went into a typhoid state, but recovered, his mental faculties, however, being shattered. During the course of the pericarditis there was a polynuclear leucocytosis, and 45 per cent. hæmoglobin.

These cases are to be differentiated from cases of severe simple chorea, in which the movements are so incessant that the patients can with difficulty be kept in bed. In simple chorea there is no delirium and there is a period of quiescence at night.

The **treatment** of chorea insaniens is symptomatic. The delirium and incessant restlessness are controlled with bromide of potassium, or sodium combined with chloral hydrate. The use of morphine is indicated in cases in which the chloral and bromides are ineffectual. Complicating endocarditis and pericarditis are treated as when primary.

FORMS OF TIC.

(*Habit Movements or Spasms.*)

This affection is mentioned in this place to emphasize the importance of sharply differentiating its forms from true Sydenham's chorea. Tic is defined by Gowers as a habitual and conscious convulsive movement of one or more of the muscles of the body, reproducing some reflex or automatic movement normal to the individual. Osler has classified the forms of tic. There is first the ordinary form, in which young people or children develop a spasm of a group of muscles, generally of the face. Children do not have the form known as idiopathic spasm of adults in which the lower extremities are involved. There is contraction of a group of facial muscles, such as the orbicularis or the muscles about the nose. There are other forms of tic in which mental disturbances and explosive utterance of words or syllables are prominent features. If the words are of an obscene character, the condition is called *coprolalia.* In other cases the patients repeat words or sentences (*echolalia*). The so-called laryngeal barks of a hysterical nature are, according to most observers, to be classified as forms of tic, and not as laryngeal chorea.

There is a fourth class, which includes those cases in which the subject before proceeding to any definite act, such as writing, feels impelled to blow on the fingers, pinch the nose, or strike the head or thorax. These actions may be regarded as harmless tricks. In another form of tic the patients feel impelled to touch objects, such as the floor or wall (delire de toucher of French writers).

RHYTHMIC MOVEMENTS OF THE HEAD ASSOCIATED WITH NYSTAGMUS.

(*Head-nodding; Spasmus Nutans; Gyrospasm.*)

Nystagmus alone is quite frequently observed in infancy and childhood.

Rhythmic movements of the head associated with nystagmus constitute an uncommon affection.

The derangement is functional and occurs in poorly nourished and rachitic infants whose nerve resistance is diminished. The majority of cases give a history of some preceding illness, in the course of which the infant has suffered from convulsions. The mothers may be of a nervous temperament. The phenomenon which at once attracts attention is a rhythmic oscillation of the head in a horizontal or vertical direction, or both. On close examination it will also be noticed that the eyes have a horizontal, vertical, or oblique form of nystagmus. Ebert, Cahen, Caillé, Gee, Hadden, and Lewi have studied these cases. Lewi reported 6 cases from my clinic. The ages of the infants ranged from three to eighteen months. The movements were augmented when the infant focussed some attractive object. The nystagmus, if not marked, may be made apparent by holding an object to the right and upward for the infant to focus. Lewi as well as Caillé found that the nystagmus ceased when the infant was blindfolded. In one case the movements continued when the infant was in the recumbent posture. The eye and head movements were not synchronous. As a rule the eye movements were the more rapid. These observers did not agree with Hadden in finding that forcible restraint of the head stopped the nystagmus. I have been accustomed to see a number of these cases yearly. Some of the infants are quite bright and well nourished. This statement agrees with that which Thomson recently made. Three-fourths of the cases are under the age of twelve months (Thomson).

The **etiology** of the affection is obscure. It is usually coincident with the period of dentition, but may appear as early as the third month. Some of the infants live in dark, squalid quarters, and the affection has been attributed to eye-strain caused by the infant's attempts to fix a light as it lies in its crib. This theory would make the affection appear similar to that frequently seen in miners (Magnus). Some of the patients that I have seen lived in well-lighted quarters.

Rachitis was present in most of my cases. Thomson's experience was similar. Henoch gives a physiological explanation of the combination of nystagmus with the rotary movements of the head, by pointing out that the root nuclei of the nerves of the muscles of the neck and throat which rotate the head are adjacent to the ocular nuclei, and that any irritation of one set of nuclei may affect the other. This explanation has been generally accepted.

Treatment.—The cases as a rule recover. They are given outdoor air, correct food, and a general course of treatment for the rachitis. Phosphorus is given as in rachitis. I have also prescribed the bromides of potassium and sodium, grains v (0.35) three times daily, but cannot say that they have had curative effects. The cases certainly improved in time. The blindfolding suggested by Caillé only stops the rhythmic movements of the head temporarily.

HYDROCEPHALUS.

(*Dropsy of the Brain.*)

Hydrocephalus or dropsy of the brain is an abnormal accumulation of fluid in the subdural space, or in the ventricles of the brain. In the former case there is external, in the latter, internal hydrocephalus. Hydrocephalus may be acute or chronic. It may also be congenital, secondary, or primary. The last-named form occurs in adult subjects (Delafield). Acute hydrocephalus is described on page 328 under the caption of Meningitis Serosa.

Congenital Internal Hydrocephalus.

The accumulation of fluid begins in utero. The quantity at birth may be small and may afterward increase. It may be large enough at birth to obstruct delivery.

Etiology.—The causes of the condition are unknown. Alcoholism, syphilis, and tuberculosis of the parents have been regarded as predisposing causes, but infants thus affected may be born of perfectly healthy parents. Sometimes several infants with this malady are born to one mother.

Morbid Anatomy.—The quantity of fluid accumulated in the ventricles varies. The fluid is perfectly clear and has a specific gravity

coprolalia. In other cases the
(*echolalia*). The so-called
are, according to most obse
not as laryngeal chorea.
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RHYTHM

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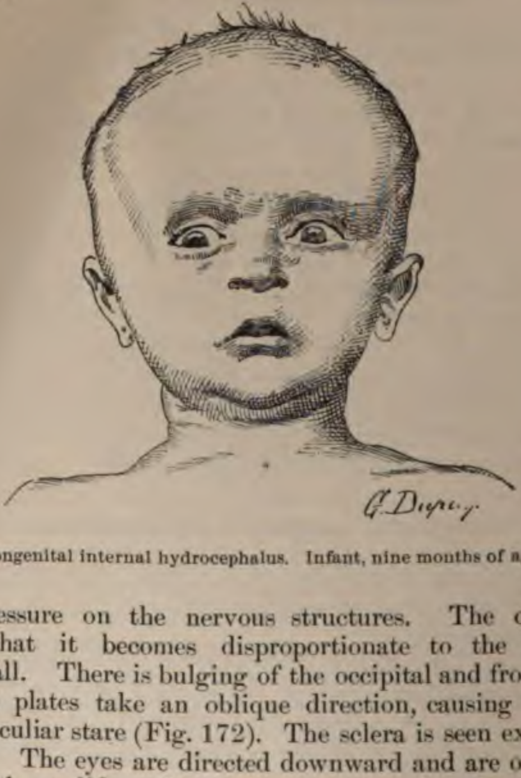
of the cerebral substance may
The convolutions may be obliter-
ganglia. The aqueduct of Sylvius is
the brain suffers most. The mem-
normal. The ependyma may be thick-

the gradually increasing size of the head
of idiocy and paralyses as a result of

FIG. 172.



Congenital internal hydrocephalus. Infant, nine months of age.

internal pressure on the nervous structures. The cranium enlarges so that it becomes disproportionate to the face, which remains small. There is bulging of the occipital and frontal regions. The orbital plates take an oblique direction, causing the eyes to assume a peculiar stare (Fig. 172). The sclera is seen exposed above the cornea. The eyes are directed downward and are only partially covered by the eyelids. The sutures are forced apart and the fontanelles are widely open. The anterior fontanelle bulges and pulsates visibly. The cranial bones may here and there show areas of thinness resembling those seen in craniotabes. The lambdoid suture and the greatest diameter is across the temples. The ain an enormous size, the child being unable to hold it The hair is scanty and dry. There may be strabismus, ontractures, and convulsions. The eyes may not be on a Blindness may result. When the disease is progressive, ocy develops. The children are very weak.

Diagnosis.—Hydrencephaloid or spurious hydrocephalus is a condition which supervenes in acute exhausting states, such as that which follows diarrhœal diseases. There is neither bulging of the fontanelles nor enlargement of the head. The fontanelle is depressed and the eyes are sunken. In certain forms of rachitis which are accompanied by craniotabes and cranial bosses over the parietal and frontal bones, there is frequently a very mild form of hydrocephalus. This condition is rarely progressive. It may be distinguished from true congenital hydrocephalus by the absence of progressive enlargement of the skull. The sutures may be patent, especially that between the parietal and frontal bones. The signs of rachitis are present elsewhere, and the children are, in contrast to the semi-idiotic subjects of hydrocephalus, very bright.

In differentiating congenital internal hydrocephalus from the external form the history is of great value. External hydrocephalus appears at birth and is not accompanied by bulging of the frontal and occipital bones. Mental deficiency is present from the outset. Late in the disease it may be impossible to distinguish between the two forms. A form of cranial syphilis is mentioned by Gowers as causing cranial enlargement, which, however, is never so marked as in congenital hydrocephalus.

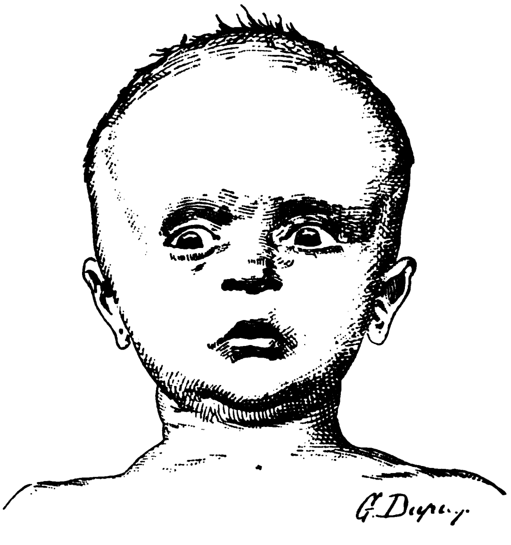
The diagnosis of congenital chronic internal hydrocephalus rests on the progressive enlargement of the cranium, the bulging in the occipital and frontal regions, and the flattening across the lambdoid suture. Acquired hydrocephalus rarely appears before the tenth month (Ireland).

It is sometimes of interest to distinguish at autopsy between the congenital and acquired forms of hydrocephalus. Meynert has shown that in congenital hydrocephalus the lateral ventricles are dilated in their long diameters; the posterior horn is dilated, so that it reaches within a few millimetres of the cranium. Acquired hydrocephalus, on the contrary, usually dilates the ventricles in their vertical and cross diameters.

Prognosis.—Hydrocephalus is one of the most fatal nervous affections. There are mild forms in which the accumulation of fluid ceases after a certain time and recovery takes place, the intelligence being either slightly weakened or normal. In some cases the enlargement continues and death ensues from marasmus. In other cases the head becomes of enormous size; the increase of fluid ceases; the fontanelles and sutures close; the unfortunate subjects have an of from 1001 to 1009. It contains a trace of albumin and sometimes urea, sodium chloride, and cholesterin. The weight may reach twenty-seven pounds. The fluid distends the lateral ventricles, the third and fifth ventricles, and the fourth to a less degree. The central canal of the cord may be dilated (Delafield). The corpus callosum is displaced upward. The thickness of the cerebral substance may be reduced to a few millimetres. The convolutions may be obliterated, as may also the basal ganglia. The aqueduct of Sylvius is dilated. The white matter of the brain suffers most. The membrane of that organ may be normal. The ependyma may be thickened and granular.

The **symptoms** are the gradually increasing size of the head and the development of idiocy and paralyses as a result of internal pressure on the nervous structures. The cranium enlarges so that it becomes disproportionate to the face, which remains small. There is bulging of the occipital and frontal regions. The orbital plates take an oblique direction, causing the eyes to assume a peculiar stare (Fig. 172). The sclera is seen exposed above the cornea. The eyes are directed downward and are only partially covered by the eyelids. The sutures are forced apart and the fontanelles are widely open. The anterior fontanelle bulges and pulsates visibly. The cranial bones may here and there show areas of thinness resembling those seen in craniotabes. The lambdoid suture

FIG. 172.



Congenital internal hydrocephalus. Infant, nine months of age.

is flattened and the greatest diameter is across the temples. The head may attain an enormous size, the child being unable to hold it upright. The hair is scanty and dry. There may be strabismus, palsies, contractures, and convulsions. The eyes may not be on a level. Blindness may result. When the disease is progressive, idiocy develops. The children are very weak.

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The **treatment** of congenital internal hydrocephalus is alone of interest to the physician. The condition is hopeless. The injection of solutions of iodine (Morton's fluid) has been tried with doubtful results. I have had 2 cases in which the ventricles were aspirated, fluid was withdrawn, and the head bandaged. The operations were performed by an expert under antiseptic precautions. In neither case was the course of the disease affected. The fluid reaccumulated. Both patients died. I have performed lumbar puncture on several cases, repeated at short intervals without permanent benefit. In one case the temperature rose to 108° F. (42.2° C.) Cheyne-Stokes respiration set in, and the patient died.

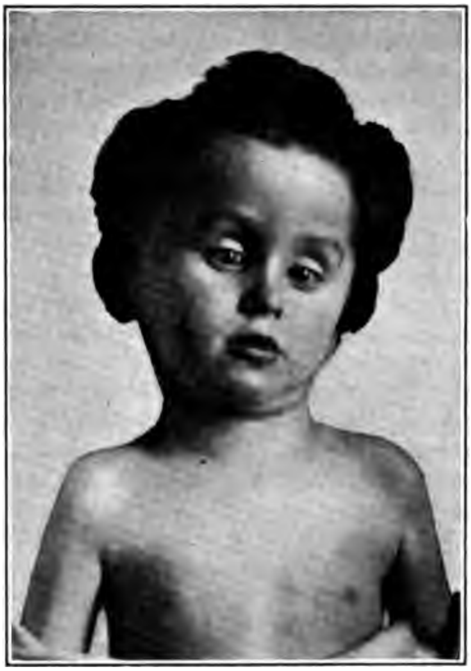
Cases in which Keen, of Philadelphia, inserted a permanent drain did not give encouraging results. Pott had an equally discouraging experience with that mode of treatment. Iodide of potassium administered internally is of doubtful value. In estimating the results of treatment, it should not be forgotten that a small percentage of cases cease to progress at a certain stage of the disease, and make a tolerably fair spontaneous recovery.

External Hydrocephalus.

External hydrocephalus may be acquired or congenital. If congenital, it follows an intra-uterine pachymeningitis or may take place because of the rudimentary state of the cerebrum (hydrocephalus anencephalique). External hydrocephalus may be acquired, in which case it follows a pachymeningitis internal hæmorrhagica or is the result of a meningitis in infancy. I have seen such cases. The congenital form of external hydrocephalus is very rare. Bokai records a case in an infant nine months of age. There was an accumulation of fluid between the dura and the pia mater. Both membranes and the falx were thickened, but there were otherwise no signs of inflammation. The infant had spastic symptoms. The diagnostic points in these cases are the uniform enlargement of the head and the bulging, especially in the temporal region. The axes of the eyes remain normal, the condition of those organs differing in that respect from that seen in internal hydrocephalus, in which they are depressed downward. There may be slight exophthalmos. In Lewis Smith's case, the axes of the eyes were normal.

In some cases of external hydrocephalus the head attains an enormous size. The disease cannot then be distinguished from the chronic internal form. In one of my cases external hydrocephalus followed meningitis. The head was uniformly large, the bulging over the temporal region being marked. The axes of the eyes were normal. The intelligence was low.

FIG. 173.



External hydrocephalus.

In some cases of external hydrocephalus there is a slight internal hydrocephalus.

AMAUROTIC IDIOCY.

(*Family Idiocy*—SACHS.)

This disease was first described by Warren Tay, an English oculist, in 1881. Among other symptoms, he noticed peculiar changes in the fundus of an infant suffering from the affection. We owe the more extensive study of the affection to the American neurologist Sachs, who described his first case in 1887, not knowing that Tay and Kingdon had previously published theirs. Sachs has collected 27 cases in the literature, his own cases being included in the number. I have published 2 cases and have since seen a number of others.

The **etiology** of the affection is still unknown. Alcoholism and syphilis do not appear to be very closely connected with its occurrence. It appears to run in families. Frequently two or more children in a family are affected.

Morbid Anatomy.—Tay-Kingdon, Sachs, and Van Giesen have studied the changes which occur in the nervous system. The first cases of Sachs were examined by Van Giesen, who found that the ganglion-cells of the cortex of the cerebrum, and especially the pyramidal cells, showed changes which indicated an arrest of development—an agenesis corticalis. There were no changes in the ganglion-cells of the cord and nuclear masses. There were degenerative areas in the white matter of the lateral tracts. Hirsch has lately examined, by new methods, the brain and cord in a case of amaurotic idiocy. He found a uniform degeneration of the ganglion-cells of the gray matter throughout the whole nervous system. There were chromatolysis and displacement of the nuclei of the ganglion-cells toward the periphery of the cell-body, with a destruction and breaking off of the dendrites and axis-cylinders. These changes were found throughout the gray matter of the brain and spinal cord. Hirsch thinks that these changes support his theory of the toxic nature of the disease. He is inclined to regard amaurotic idiocy as a form of infection originating in the intestinal canal. In a subsequent study upon cases which were in my hospital service Sachs agrees substantially with Hirsch in his anatomical findings.

The **symptoms** are divided as follows: (1) Psychical disturbances tending to complete idiocy. (2) Weakness, resulting after a time in complete paralysis. (3) A normal, diminished, or increased state of the deep reflexes. (4) Increasing blindness with pathognomonic changes in the region of the macula lutea (Tay and Kingdon's spot), with optic neuritis. (5) Marasmus.

The history of all the cases is practically the same. The infant is normal at birth. After from two to eight months, it is found to be indifferent to its surroundings. It rolls the eyes here and there. Although well nourished, it cannot sit up or hold the head upright. The head falls backward when an attempt is made to cause the infant to sit upright. Many of the infants cry constantly, at the same time making automatic facial grimaces. The lower extremities are weak and may exhibit complete paralysis (diplegia). In other cases, there may at intervals be a spastic rigidity of the lower extremities, alternating with a lax condition. Convulsions are absent or may occur occasionally. The deep reflexes may be normal or diminished. In the spastic cases they are increased. After the first year the infants become totally blind and completely idiotic. They finally become marantic, and die after the second year with the symptoms of advanced infantile atrophy. Occasionally there are nystagmus, strabismus, and hyperacusis. Deafness supervenes in many cases. The electrical contractility of the muscles may be normal or, as in one of my cases, diminished.

Ocular Changes.—The changes in the fundus of the eye described by Tay and Kingdon have been confirmed in the cases of Sachs, Koller, Heiman, and in my cases. They are invariably present at some period of the disease, but may only appear late, as in the cases of Koller. Once present, they fix the diagnosis absolutely. The appearances consist of a cherry-red spot on a diffusely white area at the region of the macula lutea. Optic neuritis is also present toward the close of the disease.

Diagnosis is not difficult after a study of the symptoms. If an infant is brought to the physician with a history of good health and intelligence up to a certain time, after which weakness and loss of interest in its surroundings set in, with inability to hold the head upright, the fundus of the eye should be examined. If Tay-Kingdon's spot is found, the diagnosis is fixed I have lately seen a number of cases in which the spastic symptoms were predominant. There were idiocy, increase of reflexes, complete or total blindness, and hyperacuity. I have watched infants with these symptoms for a long time and failed, even with expert aid, to find Tay-Kingdon's spot. In these cases there was probably a birth palsy.

The **prognosis** is invariably fatal. Of the 27 cases of Sachs, only 1 lived to the age of six years. Most of the infants die before the end of the second year

TUMORS OF THE BRAIN.

Fully 50 per cent. of the brain tumors in infancy and childhood are tuberculous; gliomata and sarcomata are next in order of frequency. Cysts are secondary to a hemorrhage or embolism. They may remain stationary for a long period, and then increase in size and cause symptoms. Males are affected twice as frequently as females; two-thirds of the cases in male subjects are cases of gliomata and tubercle. Tumors are rare in the first six months of life. The largest number occur in the first decade.

Location.—The medulla is rarely the seat of tumor. The cerebellum is most frequently involved (50 per cent. of the cases, Gerhardt, Peterson). The pars centrum ovale and basal ganglia are the parts next most frequently affected.

Etiology.—The rôle of traumatism is not clearly understood. Gliomata are due to a proliferation of the neuroglia. Tubercle and sarcomata are secondary to foci elsewhere. Carcinoma is rare. In some cases of that growth the orbit is a focus of infection.

Symptoms —Symptoms of pressure and irritation vary with the location of the tumor. A small but rapidly growing tumor will cause more pronounced symptoms than a large tumor of slow growth. Interference with the blood-supply and an increase in the quantity of fluid within the ventricles of the brain will cause the symptoms to vary.

General Symptoms.—HEADACHE.—This may in cortical and meningeal tumors be intense. It is of a boring, gnawing character, and is referred to the region of the tumor. Tumors in infants may attain great size previous to ossification of the skull. The bones of the skull are pushed apart and the sutures opened up. There is very little pain. Sleeplessness and restlessness, emaciation, and cerebral excitement are marked.

NAUSEA and VOMITING are prominent symptoms and persist for a long time. The vomiting is projectile and occurs independently of the ingestion of food.

VERTIGO is common and occurs with every change in the position of the head. It is a common symptom in tumors of the pons and cerebellum.

CONVULSIONS.—These may be localized or general. They occur when the cortex and motor areas are invaded, and eventuate in epilepsy of the Jacksonian type. In this form of epilepsy, the attack begins in the head or arm corresponding to the area of irritation, and subsequently becomes general.

OPTIC NEURITIS and optic atrophy are important symptoms of intracranial tumor, but are not always present. When tumors are situated at the base of the brain, the symptoms appear early and are due to pressure on the chiasm. Optic neuritis is either double or more pronounced in one eye.

The PULSE and RESPIRATION present no characteristic features. They show irregularities in rate. Respiration is affected only toward the close of the affection.

Symptoms Dependent on the Location of the Tumor.—CORTICAL TUMORS in or near the motor areas cause convulsive seizures, which occur from the outset. Subcortical tumors will at first cause paralysis and, as they encroach upon the cortex, convulsions. With invasion of the cortex there are, in addition to convulsions with subsequent epilepsy, intense headaches. Tubercle, glioma, and gumma occur near the surface. Cysts and sarcoma are more deeply situated.

THE FRONTAL LOBE.—The tumors situated in this region cause stupidity and other marked changes in the degree of intelligence. There will be a perversion of the sense of smell, salivation, and also the drooling seen in idiocy. If the third frontal convolution is affected, there will be motor aphasia associated with agraphia—a rare condition in childhood. Tumors of the motor area will in the earlier stages cause cortical irritation, manifested in convulsive twitchings in the parts first paralyzed. There may be slight sensory or motor disturbances in an upper extremity and an occasional twitching of the arm, forearm, or thumb.

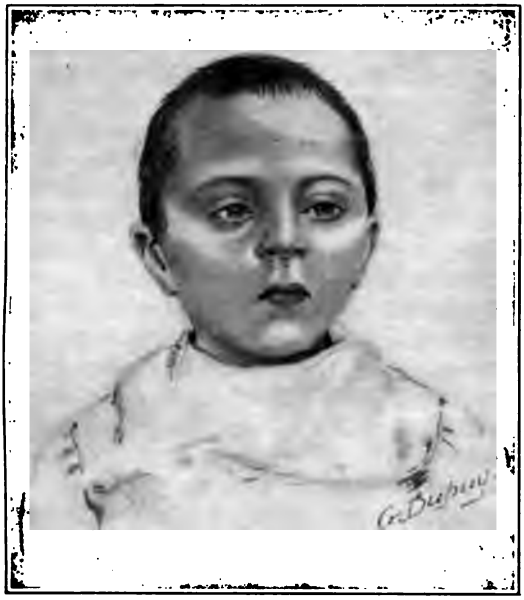
The tumors of the PARIETAL LOBE cause sensory changes in the limbs of the opposite side of the body (Dana). If the white substance is the seat of tumor, there may be hemianopsia; Wernicke's centre for conjugate movement of the eyes may be affected if the tumor is situated in the inferior part of the parietal lobe.

Tumors of the OCCIPITAL LOBE cause homonymous hemianopsia with or without epileptiform convulsions, the latter being probably due to invasion of the cortex.

Tumors of the TEMPOROSPHENOIDAL LOBE cause impairment of hearing on the side opposite to the lesion and sensory aphasia. The patient is able to speak, but cannot understand what is said or repeat spoken language.

In tumors of the GANGLIA, there is involvement of the internal capsules. There are no convulsions and none of the choreic and athetoid movement seen in cortical tumors.

FIG. 174.



Pons tumor, showing nuclear palsies. Left abducens paralysis.

Tumors of the CRUS CEREBRI cause paralysis of motion and sensation on the opposite side of the body, and oculomotor paralysis, ptosis, and paralysis of the muscles of the eyeball, except the external rectus and superior oblique. There will be paralysis of the sphincter iridis and ciliary muscle. There may be paralysis of both sides of the body, double ptosis, and double oculomotor symptoms. The majority of cases are at first unilateral, later becoming bilateral. Loss of pupillary reflex, nystagmus, and cerebellar ataxia point to involvement of the corpora quadrigemina.

Tumors of the PONS cause unilateral or bilateral symptoms. There is hemiplegia or double hemiplegia with paralysis of the cranial nerves. There is paralysis of the third, fifth, sixth, seventh, and eighth nerves of the side of the lesion, with hemiplegia of the opposite side. There may thus be paralysis of the external rectus with facial palsy and impairment of hearing on one side. If the nucleus of the sixth nerve is involved, there will be paralysis of conjugate movement of the eyes toward the side of the lesion, while if it is not affected there will be only external rectus palsy of the side of the lesion not affecting conjugate movement of the other eye.

Tumors of the MEDULLA manifest themselves in bulbar symptoms. There will be paralysis of the glossopharyngeal, vagus, spinal, accessory, and hypoglossal nerves. Thus there are unilateral or bilateral paralysis of the arms or legs, difficult deglutition, and disturbances of the respiratory movements and of cardiac action. In addition there will be spasm of the sternomastoid and trapezius muscles, and paralysis of the tongue, with atrophy, vomiting, polyuria, and glycosuria ; optic neuritis occurs early, and there is severe occipital headache. Gummata in this region are not uncommon.

Tumors of the CEREBELLUM, which are usually of the solitary tuberculous form, are the most important intracranial growths in children. There will be occipital headache, vomiting early in the disease, and cerebellar titubation due to encroachment upon the middle peduncle. Vertigo is severe. The sixth, seventh, or eighth cranial nerves may be involved. There may be bulbar symptoms. Paralysis of the external rectus is very common in these tumors. Optic neuritis may be present.

This list is by no means complete. The notes are given as concisely as possible. For further details the student is referred to the extensive special monographs on Brain Tumors.

INFANTILE CEREBRAL PALSY.

(*Spastic Hemiplegia ; Diplegia ; Paraplegia.*)

Forms.—All clinicians of note now classify these palsies with regard to the time of onset. There are three varieties—the intra-uterine or prenatal, the birth palsies, and the post-natal forms.

Brain palsy is a common disease of infancy and childhood, and has been known to occur up to the tenth year. Gowers and Osler are agreed that these palsies are most frequent in the first two years of life. They occur with equal frequency during the first and second years.

The **etiology** of the affection is still very obscure, and differs in the various forms. In the intra-uterine or prenatal form, the causal influences are especially indefinite. The influence of maternal impressions, such as fright or worry, is uncertain. Other causes frequently cited are hereditary insanity or neurotic affections, an injury or blow to the abdomen, exhausting fevers during pregnancy, pneumonia, typhoid fever, and uræmic convulsions. The rôle played in this disease by syphilis is as yet undetermined. That of alcoholism is also obscure.

The **birth palsies** have been studied by Little and McNutt. To the latter we owe the first lucid post-mortem demonstration of their cause. She published several cases under the title of Apoplexia Neonatorum. It was demonstrated in this article that in easy labors as well as in prolonged and instrumental deliveries, hemorrhages on the surface of the brain occurred and were the cause of subsequent palsies, with the resulting contractures and idiocy seen in such cases.

The Post-natal Cases.—The etiology of these cases is still a matter of discussion. When Strümpel proposed the theory of an encephalitis similar to that occurring in infantile poliomyelitis, it was for a short time accepted. Clinically this theory was founded on certain similarities between the spinal and cerebral affections. It has since been abandoned. It is found that many of the cases follow the acute infectious diseases, especially measles and scarlet fever (Gowers). Cerebral palsy may follow typhoid fever, pertussis, pneumonia, amygdalitis, cerebrospinal meningitis, gastro-enteritis, and traumatism to the skull. Infection or the presence of infectious disease cannot alone explain all the cases. The view most generally accepted is that the convulsion at the outset of the disease causes the bursting of a vessel weakened by some form of degeneration (Osler).

Morbid Anatomy.—**Prenatal Cases.**—There is porencephaly. Half a hemisphere, an entire hemisphere, or both hemispheres may be imperfectly developed. There are also certain defects in the cerebral hemisphere to which is applied the term "Agenesis Corticalis." That is to say, there is imperfect development of the cortical gray cells, particularly those of the pyramidal type. The agenesis may extend throughout all parts of the hemispheres

Birth Palsies.—The principal lesion is meningeal hemorrhage (McNutt). This may occur in areas over the cortex, or at the base of the brain. There may be a diffuse hemorrhage over the whole cortex of one hemisphere. The extravasation is, as a rule, most profuse over the motor area.

Acute Palsies.—In these, there are found embolism and thrombosis, or hemorrhage, the latter occurring mostly at an advanced age. As a result there may be atrophy of the cortex, sclerosis or cyst formations. Cysts are sometimes found later in life, there having been no previous symptoms (Gowers). They undoubtedly originate in infancy. Some authors (Gowers) state that embolism, others that hemorrhage, is the pathological condition most frequently found in cerebral palsies of acute origin. The cause of hemorrhage in these cases is still a matter of speculation. There is certainly a change in the bloodvessels, but whether it is the fatty change seen in the bloodvessels in infancy and first pointed out by von Recklinghausen, is a question. It may be that, given a vulnerable bloodvessel, heart disease or any infectious disease will predispose to hemorrhage. Cysts are likely to be found in cases in which there is idiocy.

Symptoms.—In the prenatal cases the infant is born with the disease partially or completely developed. There are cases in which no symptoms are seen at birth. They develop during the first year.

Birth Palsies.—In these cases, as in those of McNutt, there are symptoms of hemorrhage, disturbance of respiration, partial or complete loss of consciousness, and convulsions which may be general, or involve only one side of the body. The majority of infants thus affected die soon after birth; others live to exhibit various symptoms of defective development of the brain. There are paralyses more or less complete, with contractures, spastic rigidity of sets of muscles or of all the muscles, and increase of tendon reflex.

The clinical history of the very mild forms of cerebral birth palsy is characteristic. Many of the cases are mistaken for cases of asphyxia neonatorum. After a normal delivery, the infant is noticed not to breathe deeply or regularly and to appear at times to cease breathing. If roused, it cries feebly, but again relapses into a quiescent state, the breathing being irregular and shallow. It becomes slightly cyanosed, and while in this state may have repeated convulsions. In other cases the infant is born apparently well, but after twenty-four hours the respirations become shallow and increase in frequency, and convulsions appear. These cases may recover. At about the time when the infants attempt to walk they may show slight spastic symptoms in the lower extremities, and have overextension of the foot and toes as soon as any attempt is made to place the feet on the ground.

In severe cases unilateral paralysis and weakness of the muscles which support the head remain permanent. Spastic and paralytic symptoms are in some cases very marked. Athetoid movements of the extremities, with inability to grasp objects, contractures of the lower extremities, adduction of the thighs, mental defects, inability to sit upright, and oscillation in walking complete the later picture.

Acute Cerebral Palsy.—According to Gowers, the onset is acute in two-thirds of the cases occurring before the end of the second year. The condition occurs with the same frequency in the first and second years, and is as a rule primary (Gowers). Although, as has been stated, it may follow the infectious diseases, there are frequently no premonitory symptoms; the patient retires in good health and awakens with a hemiplegia. In more than half the cases the disease is ushered in with convulsions, generally unilateral. Whether these are present or not, there is unconsciousness lasting a few hours or for days, and sometimes fever and vomiting. When the child recovers the hemiplegia may be complete or the paralysis may develop slowly. The right side is more often involved (Osler).

The symptoms in order of occurrence are as follows:

Convulsions.—Coma and convulsions may be present at the outset, but may not develop until later in the disease. They are most likely to occur at the outset of the infectious diseases. If they persist into the later periods, epilepsy is likely to develop. I have seen a case in which as many as forty convulsions of an epileptiform character occurred daily. A condition of weak-mindedness or complete imbecility supervenes. Many of these cases are mistaken for true epilepsy.

Paralysis.—In all forms of cerebral palsy, there may be hemiplegia, diplegia, or paraplegia. As a rule, hemiplegia is of the right side (Osler). The facial muscles of the same side may be involved. There may be no facial paralysis. Monoplegia, especially of the leg, is rare. Diplegia is not common. After a time, contractures of the extremities occur. With these changes, there are characteristic disturbances of motion. There is the gait of the hemiplegic, slightly or markedly developed. As has previously been stated, the children may be born with contractures. A spastic paraplegia with athetosis in the upper extremities indicates the possibility that the arm may have been involved earlier in the disease. In about 20 per cent. of the cases, the face is involved at the outset. Motor aphasia is present. It is not present in birth palsies. According to Freund, hemianopsia is occasionally present.

Disturbances of sensation are rare.

Reflexes.—The deep reflexes are increased. There is no atrophy of the muscles as in infantile spinal palsies, or it is at most slight.

With the *ataxia*, there are the athetoid movements first described by Hammond. These are sometimes marked. In some cases, there are cataleptic phenomena. In all cerebral palsies of whatever origin, paralysis, rigidity of muscles, contractures, and increase of the deep reflexes are constant features.

Diagnosis.—Intra-uterine and birth palsies give a distinct history of early development. If a palsy has developed a few months after a normal labor, it is to be classed as possibly intra-uterine. Both prenatal and birth palsies are likely to be diplegic or paraplegic. As a rule there is mental deficiency. Paralysis may be complete, or, as in one of my cases, scarcely noticeable. Double athetosis is indicative of double hemiplegia, and may even take the place of paralysis. Choreiform movements are frequently mistaken for chorea. They are unilateral and combined with exaggerated reflexes and partial, slight or marked paralysis. Aphasia of cerebral palsies is motor rather than sensory. Its presence precludes the possibility of the palsy's being of prenatal or of birth origin.

The cerebral palsies are differentiated from the infantile forms of paralysis by the presence of contractures, rigidity, increase of deep reflexes, and occasionally by the presence of athetosis and choreiform movements. In recent cases the absence of atrophy will also aid in diagnosis.

Prognosis.—So far as prenatal and birth forms of palsy are concerned, no definite prediction in regard to the outcome can at first be made. Many of the cases of birth palsy die at the outset. Some escape with very slight paralysis. Others develop convulsions with subsequent epilepsy and idiocy. Contractures, diplegia, and double hemiplegia with spastic symptoms may develop. The acute cerebral forms may improve to such an extent that only slight paralysis, choreiform movements, or athetosis remain. In other cases improvement is followed by a return of the symptoms, with convulsions and epilepsy. It is estimated that fully 45 per cent. of the cerebral palsies develop epilepsy, while the diplegic forms are less likely to do so. One convulsion is apt to be followed by others, and these in time by epilepsy and mental deficiencies.

The **treatment** of cerebral palsy is ultra-conservative. Cases of birth palsy have difficulty in deglutition. Aid in keeping up the nutrition of the patient may be given by spoon-feeding or feeding with stomach-tubes (gavage). If there are convulsions, bromides in moderate doses are administered. The infant should be kept perfectly quiet. In the acute cerebral cases, if hemorrhage is suspected rest and the application of an ice-bag to the head are indicated. Subsequent convulsions are treated with bromides. The bowels are kept open with calomel. In cases in which there is slightly marked paralysis, massage and the various forms of hydrotherapy are of great utility. The faradic current has much the same effect as massage. If contractures and choreiform movements supervene, the various orthopædic appliances are of great practical utility. Where indicated, they should be used in connection with judicious tenotomy. Surgical interference has been practised in forms of epilepsy which simulate the Jacksonian type. The results are disastrous in young children, nor is permanent relief to be expected in older ones.

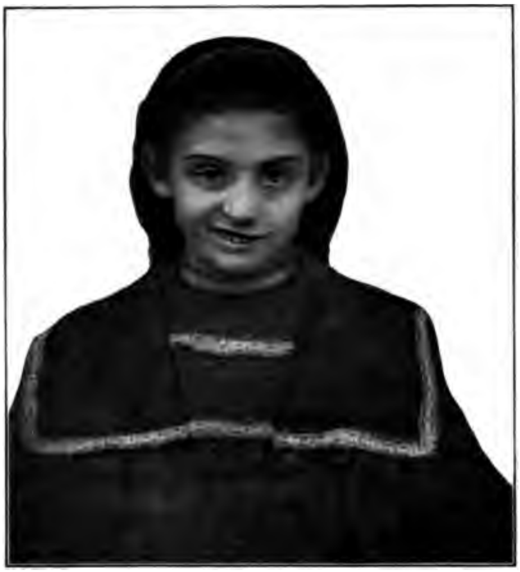
FACIAL PALSY.

(*Bell's Paralysis.*)

Paralysis of the facial nerve is quite common in infancy and childhood. As in the adult, the distribution and etiology of the paralysis vary.

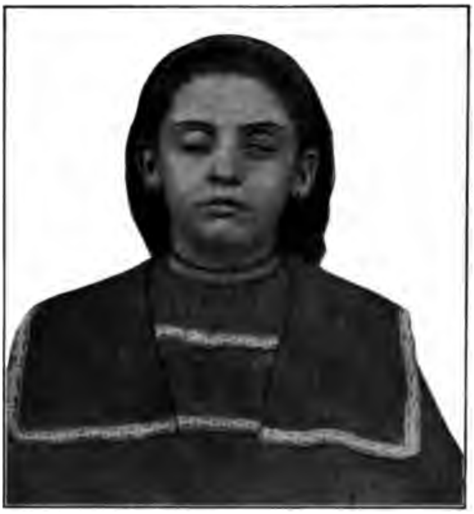
The facial paralysis observed in infants who have been delivered with forceps is a pressure paralysis. It may affect the upper or lower branches of distribution. The prognosis of this form of paralysis is, as a rule, very good. Recovery takes place after a few weeks. Some cases do not thus recover; there should therefore

FIG. 175.



Facial paralysis, left side, rheumatic form. Girl, eight years of age.

FIG. 176.



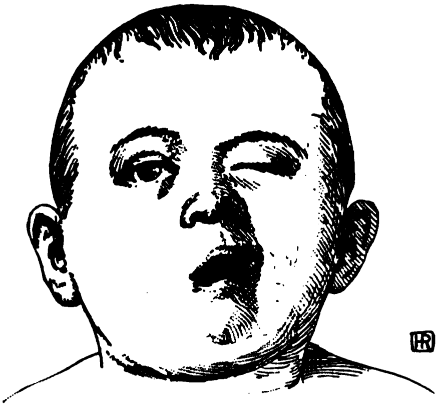
Facial paralysis, rheumatic form, showing inability to close the eye. Girl, eight years of age.

be some conservatism in prognosis. Congenital facial palsy may occur in the absence of any history of traumatism or pressure. Henoch records such a case in a boy of ten years. There was deafness on the side of the paralysis, but no history of disease of the ear.

The so-called rheumatic form of facial paralysis occurs in infants and children, but rarely does so before the third year, and most commonly between the sixth and fifteenth years. The symptoms are the same as in later life (Figs. 175 and 176).

Of greatest interest to the practitioner are the facial palsies which occur in infants and children as a result of ear disease or of inflammatory disease of the mastoid process. In infants a few months old, I have seen facial palsy due to otitis in one ear (Fig. 177). Henoch has seen cases in infants from three to five months of age. The facial nerve is affected as it passes through the Fallopian canal. Caries of the bone, pus, or swelling in the vicinity of the canal, will cause this form of paralysis. It is therefore a species of pressure paralysis. There may be no distinct collection of pus in the mastoid cells, but, when opened up, the mastoid is found to be filled with granulations. Temperature, tenderness, and redness over the mastoid should arouse suspicion.

FIG. 177.



Facial palsy complicating otitis. Infant, seven months of age.

Bokai reports a case of retropharyngeal abscess in which the facial palsy was caused by pressure on the nerve as it emerged from the stylo-mastoid foramen.

Another form of facial palsy is that seen in basilar disease of the brain. The facial palsy seen in tuberculous meningitis and sometimes in the non-tuberculous variety is of great diagnostic import. This paralysis is not always marked; it is often a very slight paresis with flattening of the facial muscles on one side and accompanied by slight widening of the palpebral fissure on the same side. In connection with this symptom, a dilatation of one pupil or slight strabismus is exceedingly significant of basilar affection. In other words, in the forms of meningitic facial palsy, the physician should be on the alert for changes in the contour of the face, since in many of these cases the patient is conscious only at intervals. In many cases, restlessness on the part of the patient will cause the slight flatness of the face or widening of the palpebral fissure to disappear. The patient should be watched unawares or when at perfect rest. The facial palsies with cerebellar tumors and tumors of the pons have been referred to in the section on Tumors.

Operative facial palsy in infants and children is likely to occur after the radical operation on the mastoid, if the operator is not a thorough anatomist. I have felt that this accident could be avoided. After an operation on the mastoid I have seen mild facial palsy, consisting of a very slight lagophthalmos with slight flattening of the facial muscles, which disappeared within twenty-four hours. It was possibly due to pressure on the nerve during the operation. Facial palsy following a mastoid operation is, as a rule, due to actual traumatism to the nerve, and to its partial or total destruction. The paralysis in such cases is permanent.

The **treatment** of facial palsy in infants and children is determined by the origin of the palsy, and is essentially the same as in the adult.

MULTIPLE NEURITIS.

This is an affection in which several or most of the peripheral nerves undergo degeneration of an acute type. The nerves affected are, as a rule, symmetrically distributed.

Etiology.—The disease may be caused by the poisonous action of drugs, such as lead, arsenic, and alcohol. It follows the infectious diseases—measles, diphtheria, typhoid fever, influenza, and malaria. In such cases the degeneration is due to the action of bacterial toxins on the peripheral nerves. Cold is said to favor the onset of the disease. In many cases, it is impossible to fix upon any definite cause.

Frequency.—If we except diphtheritic paralysis, affections of the peripheral neves are much less common in childhood than in later life. It is extremely rare in early infancy, though I have seen multiple neuritis follow measles, in which the nerves of the face, the eyes, the soft palate, the extremities, and trunk were involved in a child of fourteen months of age. When it does occur in childhood, there is a strong hereditary predisposition, or the morbific influence in the case has especial predilection for the peripheral nerves.

Morbid Anatomy.—There is an early stage during which there are hyperæmia and swelling of the sheaths of the nerves, which may be the seat of minute hemorrhages. The nuclei of the sheaths are enlarged. There is an increase of connective-tissue cells between the nerve-sheaths, and also of round and spindle-shaped cells between the nerve-fibres. The changes in the nerve-fibres are characteristic of nerve degeneration. The muscles may be the seat of parenchymatous degeneration. The striation may become indistinct. In some cases there are also interstitial changes.

The **symptoms** of multiple neuritis in children are very characteristic. After an infectious disease, the child no longer walks with a steady gait, but may stumble and fall. After a time it is noticed that the patient does not care to stand, and the mother is unable to persuade it to do so. The child cries when put on its feet, which refuse to support it. There seems to be pain connected with an attempt to stand, and also on handling and pressing the muscles. After a time the child does not sit upright, but falls back or toward one side when put in the sitting posture. It finally becomes completely paralyzed. The paralysis is progressive and symmetrical. The child does not use the hands. The feet drop forward (foot-drop) and there is a very characteristic wrist-drop. The child lies helpless in the crib, unable to move. Some of these patients cry constantly as if in pain. During this time there is good nutrition and the appetite is good. The muscles of the trunk are frequently affected as well as those of the extremities. In these cases there is a species of paralytic lordosis when the child stands or sits upright. In a few cases the muscles of the eye are affected, and in fatal cases those of the diaphragm.

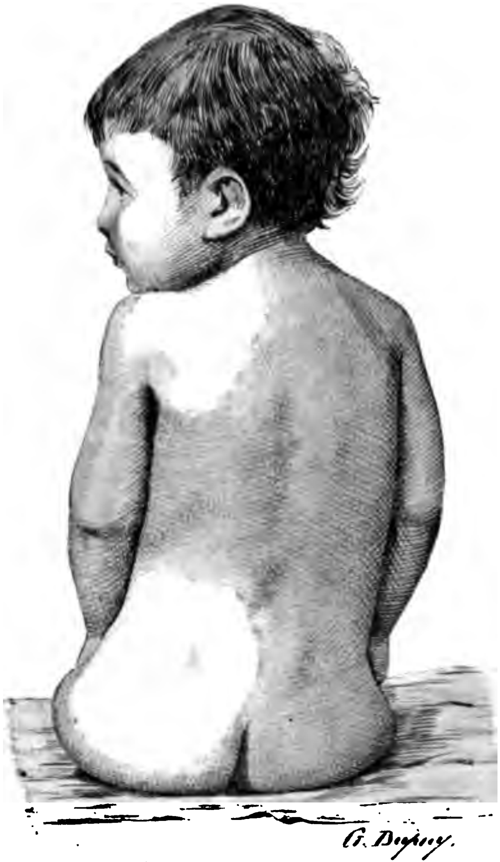
The facial and hypoglossal nerves are rarely the seat of the disease. The musculospiral and peroneal nerves seem, as in poliomyelitis, to be affected. The reflexes are diminished and finally disappear. The dorsum of the feet and hands is slightly affected with œdema.

Sensory Disturbances.—In spite of statements to the contrary, it is very difficult in children and infants to elicit exact data as to the pain or sensory changes and their distribution. I have found evidences of pain on handling the children or attempting to make them stand or sit. The patients are restless at night, and cry most of the time, and it must therefore be inferred that they have pain.

Course.—The majority of the cases make a complete or almost complete recovery. In a case which I watched very closely the reflexes were slow to return, although the child began to sit upright, then to stand, and finally to walk. The gait in walking was very peculiar. It was a sort of waddle, resembling that exhibited in congenital luxation of the hips. The boy, three years of age, finally made a complete recovery.

As a rule, the symptoms increase in severity for from four to six weeks; they then retrograde and improvement sets in. In some cases the development of symptoms is rapid, the diaphragm becomes affected, and the children die of bronchopneumonia. If the vagus is affected, death occurs through cardiac failure. Diphtheritic cases are apt to be progressive and fatal.

FIG. 178.



Multiple neuritis in a child two and one-half years of age. Shows the complete relaxation of the glutei muscles. Recovery.

Diagnosis.—If the clinical picture is studied, the diagnosis is not difficult. The complete and absolute paralysis is, in its mode of onset and its symmetrical distribution with anatomical impairment of sensation of all kinds, so peculiar that it cannot be confounded with poliomyelitis. In the cases which I have seen the muscular atrophy was also less marked than in the latter disease. The very characteristic feature of the paralysis is its flaccidity. If the child is made to sit upright, the glutei muscles flare, as it were, outside the body-line and do not retain the tonicity of the normal muscle. There is nevertheless not much atrophy of the glutei. Landry's paralysis is so rare in infancy and childhood that it need not be considered in detail.

The pain in these cases is always marked, even in young children and infants. They cry when handled, and resist all examination. In older children pressure on the nerve-trunks at their point of exposure underneath the skin, such as in the popliteal space or in the sacro-iliac groove, is exceedingly painful. The complete recovery in favorable cases without paralysis or paresis differentiates it from poliomyelitis.

The **treatment** is palliative, since the disease is not only self-limited, but also tends to spontaneous recovery. The pain is relieved and the skin kept in good condition by massage. If the child is restless, it is treated in the ordinary way. There is no specific for the affection. Electricity is not recommended by those whose experience gives weight to an opinion. If contractures result, orthopedic appliances are indicated as in other paralytic diseases.

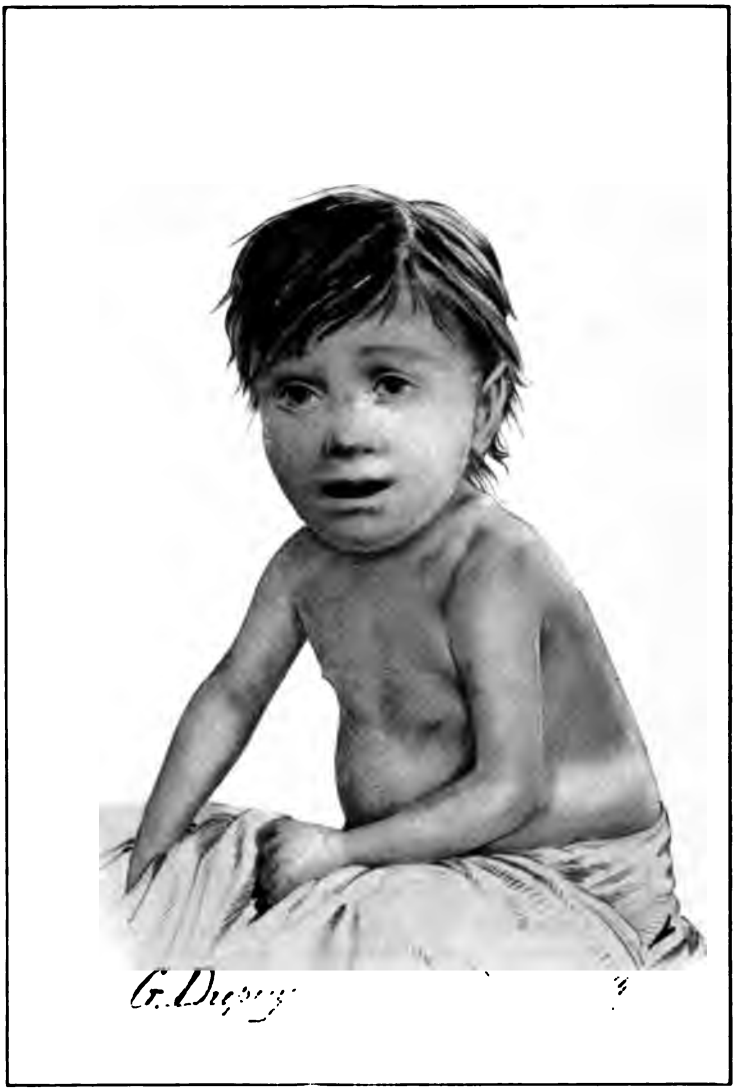
ERB'S PALSY.

(*Obstetrical Palsy.*)

This form of palsy, which occurs in infants and children as well as in adults, is due to a neuritis caused by direct traumatism either to the nerves supplying the muscles of the shoulder, or as in the newly born infant by traction or pressure on the brachial plexus (see Fig. 28). Erb showed that the point injured in these cases is the spot between the scaleni at the exit of the fifth and sixth cervical nerve roots. Duchenne, Seeligmüller, and Henoch have described these birth cases in infants. I have seen cases in older children which correspond to the adult cases.

The **symptoms** are very characteristic. There is complete paralysis of the arm on the affected side. The child, if directed to raise the arm or forearm, is unable to do so. The fingers can be moved. Infants sometimes hold the paralyzed arm with the healthy one. In a few cases there seems to be pain, caused by the drag of the paralyzed member on the shoulder. After a time there is atrophy of the deltoid and other muscles about the shoulder-joint, which causes the bony prominences to show markedly (Plate XXXII.). The atrophy sometimes comes on very rapidly. In infants and children it is impossible to reach any conclusion in regard to the intensity of pain and the disturbances of sensation.

PLATE XXXII.



Erb's Paralysis in a Child Twenty-six Months of Age. Atrophy of the deltoid, subluxation of the arm; bony prominences marked.

The cases should be differentiated from cerebral birth palsies. Apart from the electrical reaction, the absence of hemiplegia or diplegia of a spastic nature with rigidity, the absence of increased reflex, and also of convulsions, all of which are present in birth palsies, will aid in the diagnosis. Later in life it may not be possible to determine which form is present.

The **prognosis** is good, but I have seen severe cases of obstetrical palsy which failed to recover.

The **treatment** depends on the origin of the palsy. If it is obstetrical, the arm should be put in an apparatus to protect it from injury. After two weeks, friction, massage, and a mild electrical current of the faradic variety should be applied. If contractures develop later, splints should be constructed to counteract the tendency. On the whole, the management of the cases is based on the principles which govern the treatment of peripheral palsies.

HEREDITARY ATAXIA.

(*Friedreich's Disease; Hereditary Ataxic Paraplegia.*)

This is a form of ataxy which frequently affects several members of the same family. Rütimeyer and Griffith collected 233 cases which were distributed in 107 families. In 38 cases there was a direct hereditary history. In the remainder, there was a history of alcoholism, syphilis, or consanguineous marriage. Sixty-five cases of Gowers were distributed among 19 families. Thus there was an average of 3 to each family. In some families there were 10 cases. Isolated cases are rare, and occur, as a rule, only in children. The disease affects the sexes equally. Cases have occurred as early as the second year, and as late as the twenty-fourth, but are seen most frequently between the seventh and eighth years.

Symptoms.—The onset of the disease may be gradual or abrupt. The first symptom is an impairment of coördination in the lower extremities. The patient is unsteady in walking, and stands with the feet wide apart. Some patients reel when the eyes are closed more than at other times. In other cases Romberg's symptom is absent. The feet show the peculiar deformity of pes cavus. The instep is high and the toes overextended. The movements of the arms next become ataxic. The speech becomes slow and halting. Jerking, nodding movements of the head set in. Irritability of muscle is absent from the beginning. The deep reflexes may be present at first, but finally disappear as in true tabes.

Nystagmus is usually present, and may be a very early symptom, appearing simultaneously with the ataxic symptoms. The symptoms connected with the speech may come on very late in the disease.

Optic atrophy is never present, and the Argyll-Robertson pupil of tabes is absent.

Sensory disturbances, such as shooting pains, are rare, but may occur. There is no tendency to trophic joint-affection as in tabes. The sphincters are normal.

Muscular power, although normal at first, diminishes as the disease progresses. There is atrophy of muscle. Spinal curvatures, talipes equinus, and equinovarus result. The loss of muscular power is sometimes limited to the lower extremities.

The mental condition is generally affected. The children are slow at school. Imbecility has been recorded (Gowers).

Course.—Once inaugurated, the disease is progressive, but it may remain stationary at any stage for some years. The duration is extended over years. Gowers gives the period as ten to twelve years. The patients finally become bedridden, and, as a rule, die from intercurrent disease. The anatomical changes have not as yet been completely classified. This is due to the fact that in certain forms of hereditary ataxia resembling Friedreich's disease, Marie and Hoffmann have described changes other than those found in typical cases of that affection. The changes in Friedreich's disease consist in a diminution in the transverse diameter of the cord and a sclerosis of the posterior and lateral columns, involving the pyramidal tracts. The neuroglia and vessels of the tracts are involved; whether this is due to an arrest of development of a congenital nature has not been determined.

Differential Diagnosis.—The disease should be differentiated from true tabes. In the latter, there are the Argyll-Robertson pupil and optic neuritis, the visceral crises and shooting pains, but neither head-nodding nor nystagmus. The lack of intelligence and the family history are characteristic of Friedreich's disease.

Prognosis and Treatment.—There is no cure for the affection. The treatment is designed to relieve the symptoms.

ACUTE ATROPHIC PARALYSIS.

(*Acute Anterior Poliomyelitis; Infantile Paralysis; Essential Paraylsis of Children.*)

This is the most common form of paralysis in infants and children. It is a disease characterized by loss of voluntary power, taking place within a few hours or days. Some of the paralyzed muscles recover; others undergo atrophy.

Occurrence.—There is doubt as to whether the disease occurs in fœtal life. Duchenne has reported a case at the twelfth day. Such early cases are apt to be cases of hemorrhage rather than of poliomyelitis. The majority of cases occurring during the first year develop after the sixth month, and three-fifths of the cases before the tenth year are found to occur during the first three years. It affects the sexes equally.

The **etiology** of the disease is still uncertain. Cases occasionally occur after exposure to cold, and after a traumatism or a psychical disturbance, such as fright. The relationship between these predisposing elements and the disease is probably no more intimate than is the case in other affections.

This disease is very frequent in the period of dentition, but since dentition has been regarded as a predisposing cause in most diseases, no close relationship is evident. It has been shown by Medin, Strümpel, and Zuppert that many of the cases of poliomyelitis occur after or during the course of the infectious diseases, especially measles, scarlet fever, and typhoid fever. It is probable that some toxin acting through the blood and blood-vessels causes the degeneration in the cord characteristic of the disease. It is also probable that the epidemic occurrence of the affection is due to its infectious nature. Medin, Pasteur, Sinkler, Putnam, Chapin, and Zuppert have reported epidemics.

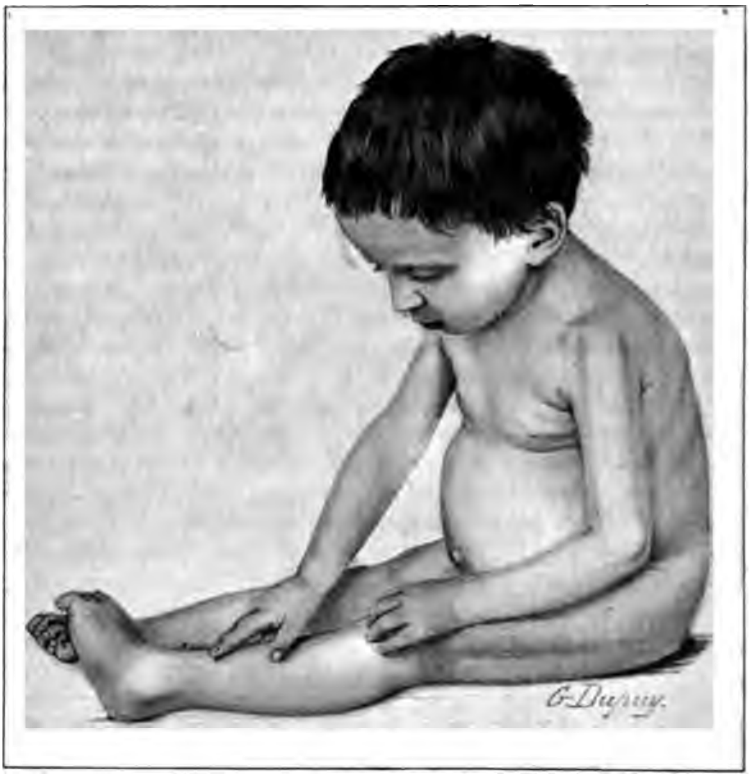
Symptoms.—There are four distinct periods in the development of the symptomatology: The period of onset, in which the symptoms resemble those of an infectious disease; the period of paralysis; the period of retrogression, in which some of the paralyzed parts recover while others remain permanently paralyzed; and finally, the chronic state, in which there are permanent paralysis and atrophy.

The Onset.—This is always acute. In most cases there are fever, vomiting, and diarrhœa. The fever may be slight (100 °F., 37.7° C.) or may mount to 104° F. (40° C.).

Complete paralysis sets in after these symptoms have continued for a few hours or days. In other cases the paralysis first attracts attention, and is followed by fever and constitutional disturbances persisting for days. General convulsions may usher in the disease, and be followed by coma lasting for days. At the termination of the coma, the patient is found to be paralyzed. The onset sometimes resembles that of cerebrospinal meningitis. There are headache, vomiting, fever, and rigidity, the paralysis becoming apparent after the subsidence of these symptoms. Other cases have absolutely no premonitary symptoms. The patient goes to bed in health, but in the morning is found to be paralyzed. Patients sometimes suddenly fall, and on being raised up are found to be paralyzed. These are probably cases of acute spinal hemorrhage. In older children pain in the course of the nerves may usher in the paralysis. Pains in the joints and back may succeed the paralysis. Such cases closely resemble those of peripheral neuritis.

The Paralysis.—The paralysis consists of a loss of power, which is complete in two or three limbs or in parts of extremities. Seeligmüller found the relative frequency of involvement to be as follows: the right lower extremity, the left lower extremity, the right upper extremity, and the left upper extremity, in the order named. All four limbs may be involved, or only a hand and a leg. If all four limbs are at first involved, there is weakness of the back. The patient cannot sit upright or hold the head erect. The cranial nerves escape, except in very rare cases, in which degeneration or inflammation involves the medulla and its nuclei. There may be symptoms which simulate those of bulbar paralysis.

FIG. 179.



Acute atrophic paralysis involving the left upper and lower extremities.

Different sets of muscles may be involved. After the first onset of the paralysis, some of the muscles may recover. Thus a child who has been unable to sit up or move the arms will recover the power to do so. In such cases one leg only may remain permanently paralyzed.

Paralysis may develop slowly in the course of one or two weeks. After that time it comes to a standstill. In a period of from one to three months either recovery will take place or the paralysis will be complete with accompanying atrophy.

Atrophy in the paralyzed muscle is very characteristic of the disease. It may be seen as early as the first week. Accompanying it, and appearing from the fifth to the seventh day, is the reaction of degeneration in the paralyzed muscle and nerve. The faradic and galvanic irritability of nerve and muscle are increased for the first two days. They then rapidly diminish, the former disappearing completely. The galvanic irritability remains increased for from two to six months; it then diminishes, and if the paralysis is permanent, disappears at the end of one or two years. In rare cases all electrical irritability disappears from the onset. In others the faradic irritability in certain fibres and muscles returns after from six to twelve months. These muscles may partially recover, but remain atrophied and weak. There is usually no loss of sensation, but if it does occur, there is incontinence of urine. Reflex at the patellar tendon is lost and myotonic irritability is either lost or diminished. In cervical disease of the cord, or when only the posterior tibial muscle, or the muscles of the feet are paralyzed, the tendon reflex at the knee is present. In rare cases, the inflammation may spread from the anterior horns to the lateral columns. The lower extremities may then be paralyzed but not atrophied, and clonus may be present.

Growth of bone is retarded, and one foot may after a time become shorter than the other. The joints become the seat of subluxations through the laxity of the muscle and lack of support. The articular ends of the bones are not held in apposition. Through the shortening of some muscles and the traction of others there will result various forms of talipes. The muscles in front of the tibia are affected more than those of the calf. The extensors of the thigh are more frequently paralyzed than the flexors.

The muscles of the whole arm may be paralyzed, or, as in Erb's paralysis, only those of the deltoid group. The serratus, the pectoralis, the muscles of the back and neck, and the diaphragm may all be affected.

Course.—The mildest cases rarely make a complete recovery. Death is very uncommon and occurs only in the early stages. It may supervene within two weeks from general paralysis or cerebral disturbance. Relapses are rare, second attacks unknown.

Sequelæ.—A cord which has once been the seat of this disease is naturally susceptible. Gowers states that he has seen chronic disease of the cord supervene later in life. Progressive muscular atrophy or lateral sclerosis may at some later time appear in the cord.

The **prognosis** of acute atrophic paralysis is good as to life. As to the outcome of the paralysis, a prediction can be made only when all the muscles which show faradic irritability have recovered. Some children who in the second stage have shown complete paresis or paralysis from the cervical region down, gradually regain power in all of the affected muscles, only one limb or part of a limb being permanently affected. As a rule those parts, which after a week respond to faradism, will recover.

Diagnosis.—At the onset, the case should be distinguished from one of the infectious diseases. Since the mode of onset is much the same, it is best, as in those diseases, to defer making the diagnosis until the initial symptoms have passed and the paralysis appears. When the paralysis is fully developed, it should be differentiated from forms of cerebral palsy. This in the majority of cases is difficult. The characteristic atrophy, the complete paralysis, the loss of knee-jerk, and the absence of contractures will all be of service. Those cases in which pain in the course of the nerves is present at the onset, should be distinguished from cases of multiple peripheral neuritis. Time and study of the cases will make this possible. In those forms of poliomyelitis in young infants, in which the muscles of the deltoid group are affected, Erb's traumatic form of shoulder paralysis should be excluded. Some cases closely resemble this form of paralysis. If the paralysis occurs immediately after birth and follows traction on the arms, poliomyelitis may be excluded. If the paralysis occurs after the sixth month, the diagnosis, in the absence of any traumatic history, should be that of poliomyelitis.

Morbid Anatomy.—The theory of Charcot, that anterior poliomyelitis is a primary degeneration of the ganglion-cells in the anterior horns of the gray matter in the cord, has given way to the belief that there is severe inflammation superinduced by some toxic agent circulating in the blood. The change begins in a degeneration of the bloodvessels of the anterior median fissure. There is proliferation of the endothelial lining of these vessels. The inflammatory process extends to the surrounding neuroglia and the ganglion-cells supplied by those vessels in the anterior horns of gray matter. In severe cases, the motor nuclei of the medulla may be involved. There may be inflammatory exudation and hemorrhage. In recent cases, the ganglion-cells show granular swelling, vacuole formation, hyaline changes, disintegration, and atrophy. After months, there is paucity of ganglion-cells in the region corresponding to the paralyzed members. They are seen in a few groups in the anterior horns or may be entirely wanting. Outside of the affected area, there may be a diminution of the number of ganglion-cells throughout the whole cord. The nerve-fibres corresponding to the ganglion-cells which have disappeared are also wanting. There may be no marked change in the glia tissue, in the transverse section of the anterior horns, and in the general configuration of the transverse section of the cord. After a time, however, there will be sclerosis and atrophy of the affected horn. The sclerosis may affect the white columns. The anterior horns and corresponding white substance may be transformed into a glia tissue resembling gelatin, the spaces containing fluid granules and disintegrated nerve-tissue

(Ziegler). All these changes point to permanent injury to the spino-muscular neuron, the ganglion-cell of the anterior horn, and its nerve-fibre.

The **treatment** of anterior poliomyelitis is symptomatic. In the stage of onset, perfect rest and quiet are indicated, and a few remedies to meet the symptoms. The bowels are kept open with calomel. Bromides are used if the patient is restless. Ice applied to the nape of the neck or to the head, as in cerebral disease, is useful if there are cerebral symptoms, such as headaches. Ergot has been given to act on the blood-supply of the cord, but is of doubtful value. After paralysis is established and atrophy has made its appearance, massage of the affected muscles, and electricity, especially of the galvanic form, are indicated. Later, in the chronic stage, much can be done for the sufferers by orthopedic appliances, such as braces and splints. If there is contraction of opposing muscles, tenotomy should be resorted to. In cases in which the joints have become the seat of luxation, arthrodesis has been practised by surgeons with good results in increasing the power of the affected limbs.

THE JUVENILE FORM OF PROGRESSIVE MUSCULAR ATROPHY (ERB'S TYPE).

This disease is characterized by a weakness and progressive wasting of certain muscles. It begins in childhood or early youth, and involves, as a rule, the shoulder-girdle, the upper arm and pelvic girdle, and the thigh and back. The muscles of the forearm and leg remain for a time intact. This atrophy may be associated with true hypertrophy or pseudohypertrophy of some muscle. The pectoralis, the trapezii, the latissimi dorsi, the serrati, the rhomboids, the upper arm muscles and supraspinators, are apt to be wasted. The deltoids, supraspinati, and infraspinati may be normal or hypertrophied for a time. There are no fibrillar contractions, no disturbances of sensation, and no reactions of degeneration and visceral disturbances.

THE LANDOUZY OR DEJERINE TYPE OF THE FACIOSCAPULO HUMERAL FORM OF MUSCULAR ATROPHY.

This form in no way differs clinically or pathologically from the juvenile form of muscular atrophy. Authors include in this class all cases in which the atrophy begins in early life, as a rule, in the muscles of the face. The patients have a peculiar expression—so-called "facies myopathique." The lips are thickened ("bouche de tapir" or tapir mouth). The shoulders later become atrophied. The supraspinati, infraspinati, and the flexors of the hands and fingers remain normal, as do the muscles of deglutition, mastication, respiration, and the laryngeal and ocular muscles. There are no fibrillary twitchings. The spinal forms of progressive muscular atrophy differ from primary dystrophy in that the onset of the latter affection is in the upper extremities. The disease is not hereditary, and fibrillary twitchings and electrical reactions of degeneration are absent.

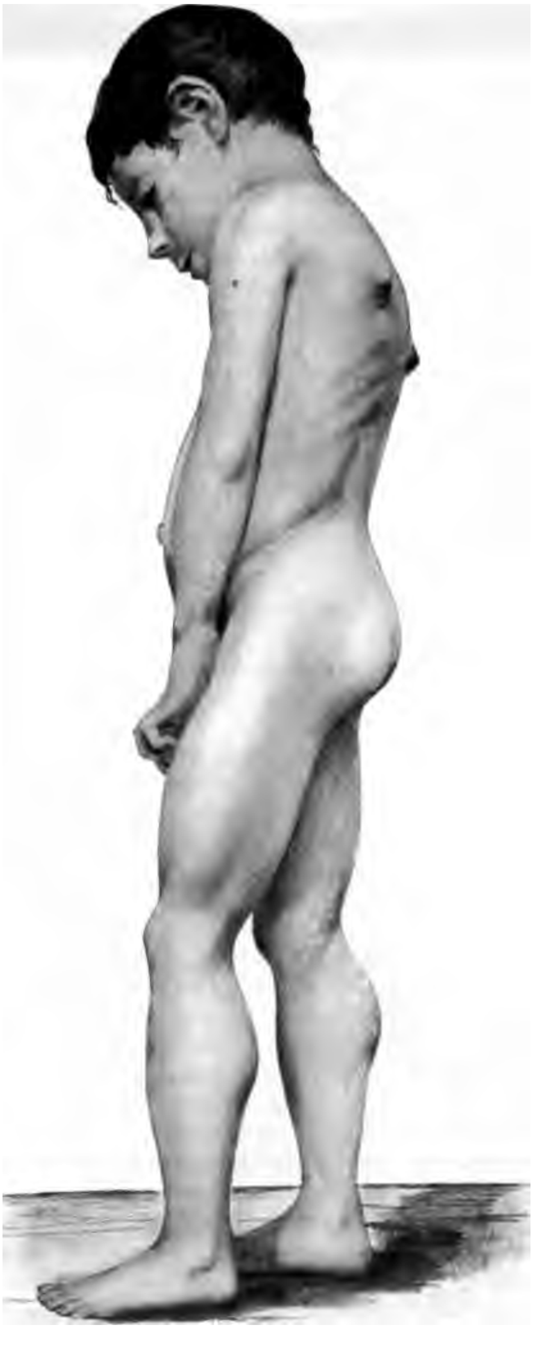
Both these forms are probably clinical varieties of the pseudohypertrophic form of paralysis.

PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

This disease is characterized by a progressive change in the size of many of the muscles of the body and by a diminution of their power. It was described by Duchenne in 1861. Since then the most notable work on the subject has been done by Gowers, of England, and Sachs, of this country. The male sex is more frequently affected than the female. From two to eight members of the same family are often affected. Isolated cases are uncommon. The disease frequently affects the members of one sex in a family group. It is congenital but not hereditary. The antecedent cases, if there are such, can usually be traced on the mother's side of the family. The mother may be herself unaffected. Intemperance does not seem to exert any influence on the occurrence. Gowers notes that frequent marriage of parties closely related tends to predispose to the development of the disease in the children. In one-third of the cases the disease appears when the child begins to walk, and in children who are late in learning. It may manifest itself in the mid-period of childhood. In another third of the cases the children are in apparently good health until the fourth or sixth year. Three-fourths of the cases show symptoms of the disease before the tenth year. The disease may not manifest itself until after puberty, and may only be noticed during convalescence from some intercurrent acute disease.

The **symptoms** are impairment of power and change in the form of groups of muscles or of single muscles. The impairment of power is at first not very apparent. The muscles of the calves enlarge, and show a very characteristic and significant hypertrophy. Mothers are at first pleased with what appears to be muscular development of the children (Gowers). It is then noticed that although the muscles of the calves and glutei are large, the children are easily fatigued in mounting stairs. They fall easily and rise

PLATE XXXIII.



Pseudohypertrophic Paralysis in a Boy Eight Years of Age. Hypertrophy of the infraspinati well shown; also atrophy of the muscles of the thorax and hypertrophy of the glutei and the muscles of the lower extremity.

with difficulty. This loss of power is at first interpreted as weakness, but when it is found to be progressive the children are brought to the physician. The gait becomes pronouncedly oscillating. The body is inclined so that the centre of gravity is brought successively over each foot. In trying to rise from the ground the patient places a hand on each knee in a very characteristic fashion. By grasping the thighs and throwing back the weight of the trunk, the patient helps himself into the erect posture. The weakness of the muscles finally becomes extreme. The patients can neither stand, walk, nor sit upright. They become bedridden. In the early stage, the muscles of the trunk may be normal, small, or atrophied, and those of the lower extremities much enlarged. Single muscles or groups of muscles of the arm and forearm may be enlarged (Plate XXXIII.). Finally, as the atrophy and weakness increase, there are contractures and distortions of the extremities and trunk. Equinus, lordosis, and lateral curvature are very marked. The knee may become fixed and distorted by contractures. The muscles most frequently affected in the beginning are those of the calves of the legs. These sometimes attain an enormous size. Those of the anterior part of the leg are not so much enlarged. The flexors of the knee commonly escape. The glutei and lumbar muscles are enlarged. The infraspinatus muscle is frequently enlarged, and stands out prominently; it is often mistaken for the lower edge of the scapula. The deltoid is often large; the serratus and the pectoralis are rarely affected. The triceps and biceps are frequently large, but often only in parts. The muscles of the forearm suffer only in a minority of cases. The intrinsic muscles of the hand are never affected. In that respect the disease is sharply distinguished from atrophies of spinal origin. The muscles of the neck are, with the exception of the clavicular portion of the sternomastoid, rarely affected. All the muscles affected are weakened, the smaller and atrophied muscles more so than the others. There is reason to believe that many muscles not visible are much affected.

Electrical Reaction.—This is altered when weakness sets in. The electrical contractility to galvanic and faradic stimulus finally disappears.

Reflexes.—The knee-jerk is at first normal. It later diminishes and finally disappears. It is never increased in a pure case. In one case in my hospital service there were increased reflex at the knee and foot-clonus. This case gave a history of a blow across the back. Sachs, with whom I saw the case, suspected a complicating myelitis of the cord.

Sensation is unaffected and the sphincters remain normal.

The **course** of the affection is prolonged and tedious. The disease is progressive. It may be ten or fourteen years before the patients succumb. They die of some intercurrent disease. If the disease appears after puberty, the course is slower than in cases in which the first symptoms are noted in early childhood.

Varieties.—There are cases in which only one muscle or group of muscles of the extremities is enlarged, the others being small or normal in size. There are other cases in which all the muscles are small and waste progressively.

Complications.—Chorea, poliomyelitis, myelitis, mental deficiencies, and epilepsy may complicate the affection.

Morbid Anatomy.—The gray matter of the cord and the nerves are normal in appearance. There may be slight hemorrhages. The neuroglia-cells have sometimes been found to be increased. The disease is, however, primarily one of the muscle-tissue. The muscles are pale-yellow. They are replaced mainly by fat and connective tissue. The muscle-fibre is narrower than is normal, although in advanced cases the residual muscle-fibre may retain its transverse striation. Where the muscle-fibre is narrow it becomes granular or is the seat of fatty or waxy degeneration and vacuolization. Empty sarcolemma-sheaths are seen.

The **diagnosis** is made from the progressive weakness, the gait, and the mode of rising from the recumbent position. The peculiar enlargement of the muscles of the calf and infraspinatus, the atrophy of the latissimus dorsi and lower part of the pectoralis, and the immunity of the intrinsic muscles of the hand are characteristic. In the stage of contracture, this disease differs from congenital spastic paraplegia in that there is no increase of deep reflexes.

The **prognosis** in children is grave. The affection is progressive.

Treatment.—Much can be done for the patients by means of massage and electricity. In the stage of contractures, while there is still power, relief can be secured by tenotomy.

DEFORMITIES OF THE SKULL AND SPINAL CANAL.

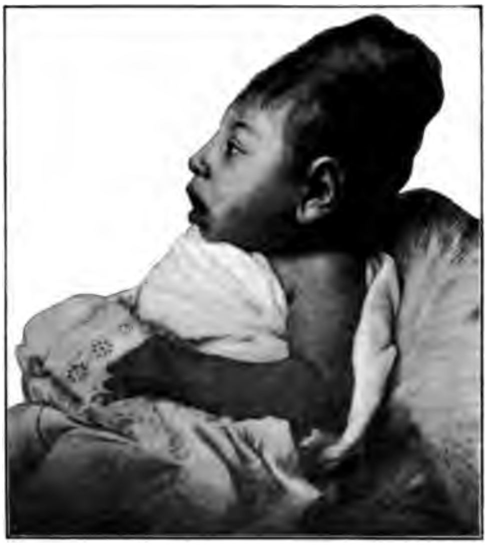
These deformities do not strictly belong to the diseases of infancy and childhood. Only the forms most commonly met are here considered.

The faulty closure of the spinal canal causes a deformity called rachischisis or spina bifida. If the defect involves the spinal canal in its whole extent, there is rachischisis totalis. The vertebræ form a shallow canal in which lies the rudimentary spinal cord covered with a thin membrane. If the defect of the bony canal is only partial, there being a sac-like protrusion of the cord and its membrane, there is said to be a rachischisis cystica or spina bifida cystica or rachicele.

Faulty development of the cranial bones with rudimentary brain is called cranioschisis (Fig. 180). If with the cranial defects there are defects of the bony vertebral canal, there is said to be craniorachischisis.

If there are only partial defects in the cranial bones, with sacculated protrusion of the membranes of the brain (pia and arachnoid), with fluid in the sac, there is a meningocele. Meningo-encephalocele is a sac containing in addition the brain-substance. Encephalocele is a hernia of the brain and pia, no fluid being present in the sac.

FIG. 180.



Cranioschisis. Deficiency of the frontal, parietal, and most of the occipital bones. Protrusion of the cranial contents in shape of a sac covered by hair and scalp, and containing fluid and brain substance. Blindness; idiocy.

Spina Bifida.

Spina bifida or hydrorrhachis is a congenital deficiency in the vertebral laminæ, through which the cord and its membranes protrude in the form of a sac containing fluid. The deformity is most frequently seen in the dorsolumbar, dorsosacral, and cervical portions of the vertebral canal. It rarely occurs in the middorsal region. It is generally single. It may occur both in the neck and in the lumbar region.

The tumor may be small and only indicated by a fissure, or may, as in Broca's case, attain a circumference of 62 cm. It may be flat or pedunculated. The latter form is uncommon. The surface of the tumor may be smooth or lobulated and uneven. The lobulated forms indicate divisions in the interior of the sac. The skin covering the sac may be very thin or glistening. It may burst during delivery, may be thick and vascular, or covered with cicatrices and granulating ulcers. In some tumors the subcutaneous tissue can be made out; in others the skin is atrophic. In rare cases the tumor is composed of a mass of mucous tissue situated between the skin and dura mater. In the interior of this mass there is a small cavity (Kirmisson). Von Recklinghausen and Muscatello have demonstrated that the statement that the sac of the spina bifida is lined with dura mater is incorrect. Hildebrandt has, however, found cases in which the dura lined the sac. The pia and arachnoid line the sac. The fluid in the sac is serous and colorless or lemon-colored. It is alkaline in reaction, rich in salts, and contains sugar. If inflammation is present, blood is found in the sac. The fluid is either outside the cord or in the central canal (Virchow).

Spina bifida is, with reference to the nature of the contents of the sac, divided into three forms:

(*a*) Myelomeningocele, in which the fluid in the sac is situated between the cord and its membranes.

(*b*) Meningocele spinalis, in which the inner surface of the sac is formed by the arachnoid and pia mater.

(*c*) Myelocystocele, in which the fluid is situated in the central canal of the cord.

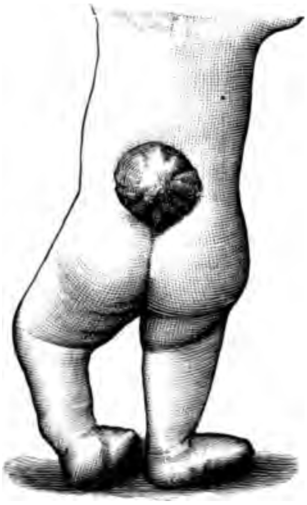
The **myelomeningocele** forms a broad but not very prominent tumor, which may be found in the lumbosacral, cervical, thoracic, or sacral regions. At its base the tumor is reddish, and is covered with fine, long hairs. This zone is from 1 to 1½ cm. broad. In the centre of the tumor there is a reddish-brown velvety vascular area, the remains of the medullary vascular zone. The sac is formed of arachnoid and pia mater. Its interior is crossed by nerve-trunks. The cord is drawn outward and some nerves may arise from the prolongations of the cord. Accordingly, there is an accumulation of fluid in the meninges (hydromeningocele), with an accompanying hernia of the cord (myelocele).

Meningocele spinalis is the rarest form of spina bifida. The sac is composed of pia and arachnoid. The latter may be much thickened. The opening into the vertebral canal if large may allow hernia of the cord. If the tumor is situated in the sacral region, the interior of the sac may contain the nerves of cauda equina.

Myelocystocele, hydromyelocele, or **syringomyelocele,** is that form of spina bifida in which there is a dilatation of the central canal of the cord. The dura is lacking in the sac, which is lined with cylindrical epithelium. The spinal cord in part of its extent may be found in the sac, or may be found on the exterior wall of the sac and end there. It may break up into several bundles. In the interior the spinal nerves form a series of loops with their convexities posteriorly. They may return into the vertebral canal or may end in the sac. Spina bifida is a primary agenesis. The growth of the sac is due to inflammatory processes.

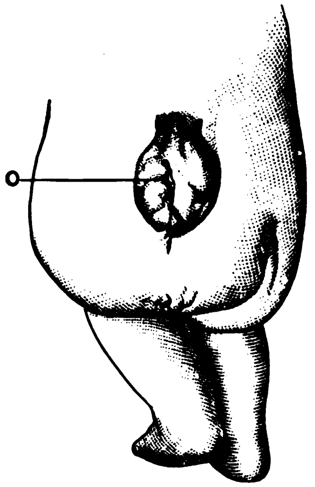
Symptoms.—The tumor is the chief physical sign. It is situated in the median line or may be at one side. It is round or elliptical and covered with thinned or thickened skin (Figs. 181 and 182). In the centre of the myelocystocele is a depression which gives the tumor a tomato-like appearance. The tumor may be soft, hard, or fluctuating. The defective vertebral laminæ may be discerned on palpation. The tumor enlarges and becomes tense when the patient assumes the upright posture, cries, or exerts himself. When the patient takes the recumbent posture it becomes smaller. It also does so at each inspiration.

FIG. 181.



Spherical form of spina bifida lumbalis.

FIG. 182.



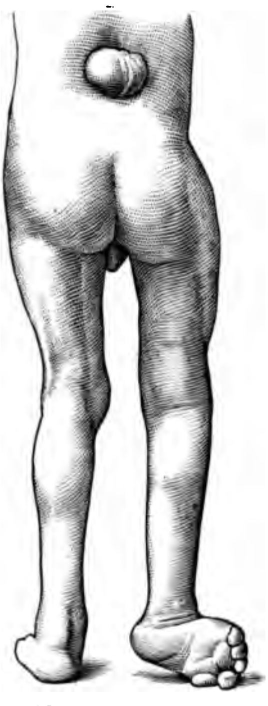
Elliptical form of spina bifida, with fistulous opening (o) into the vertebral canal.

In some cases the functions of the individual are normal. In others, the mobility and sensibility of the lower extremities are affected. Deformities of the foot similar to those seen in infantile paralysis are sometimes present. There may be incontinence of urine and feces. There are sometimes trophic disturbances, such as perforating ulcers. These are of value in the diagnosis of lumbar tumors which are apparently lipomatous in their nature and are covered with hair (Kirmisson). In such tumors, disturbances of sensibility occurring with perforating ulcers and deformity and atrophy of a lower extremity are significant of spina bifida.

Course.—Spina bifida if left to itself may grow to a large size, may burst or ulcerate, and cause death by pyogenic infection of the meninges and cord tissue. In other cases a lineal ulcer discharges fluid and closes up several times in succession. In some cases of spina bifida the tumors remain stationary in size until late in adult life. In rare cases spontaneous cure results by inflammation of the pedicle of a pedunculated spina bifida.

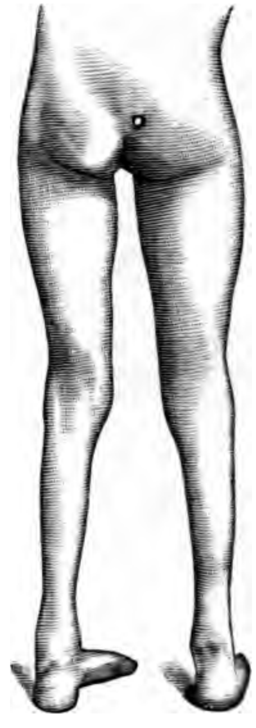
The **diagnosis** of spina bifida is not difficult if what has been detailed of the anatomy and symptomatology is borne in mind. Muscatello gives the following characteristics of the various forms:

FIG. 183.



Spina bifida lumbalis, with pes valgus on the right side; also congenital subluxation of the hip.

FIG. 184.



Spina bifida occulta pes calcaneovalgus on the right side; pes equinovarus on the left side.[1]

In **myelocystocele** there is a round tumor with a wide base. The tumor is lumbosacral, elastic, translucent, and fluctuating, and does not diminish on pressure. Pressure causes tenseness of the fontanelle. There may be scoliosis, lordosis, abdominovesical fissure, and deformity of the foot.

In **myelomeningocele** there is a flat, soft, elastic tumor, either lumbar, sacral, cervical, or thoracic. It may be complicated by umbilical hernia, paralysis of the extremities and bladder, and deformity of the foot.

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[1] Figs. 181–184 are taken from Kirmisson.

In **meningocele** there is a sacral pedunculated translucent tumor, but no disturbances of mobility or sensibility.

Of considerable interest is the form called spina bifida occulta (Fig. 184). In these cases there may be no tumor, the seat of the deformity being indicated by a depression or dimple. In other cases, as in that shown in the illustration from Kirmisson, there is a small tumor of doughy consistence on one of the gluteal folds. The tumor may present an umbilication. Spina bifida occulta should be suspected in cases in which abnormal sacral depressions or tumors occur in connection with clubfoot deformities or congenital incontinence of urine or feces, or of both.

The **treatment** of spina bifida belongs to the domain of surgery. The treatment by injections of Morton's fluid (2 per cent. of iodine, 6 per cent. of potassium iodide in glycerin) has been abandoned in favor of excision of the sac.

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SECTION XIV.

DISEASES OF THE SKIN.

THE skin of the infant is exceedingly delicate in structure. After birth there is a physiological condition of desquamation, as a result of which the skin is very sensitive to a traumatism which in older children would be considered slight. In the newly born infant, such is the delicacy of the structure of the skin that infection may occur when no lesion of continuity is apparent (cryptogenic). A rapid examination of the skin is the first step in making a full physical examination of an infant or child. The surface is first inspected from a distance, the color and the presence or absence of an eruption being noted. It is of the first importance to decide whether an eruption is acute or connected with constitutional taint (syphilis). An eczema may in a syphilitic infant have certain characteristic variations of color which will at once lead the examiner to suspect constitutional disease. A familiarity with acute eruptions (exanthematic) is essential. These must be diagnosed or excluded before any treatment can be inaugurated. Forms of œdema must be differentiated from sclerema and myxœdema, and indurations of the skin from elevations. A papule may be elevated but not indurated. Since the skin of infants and children is exceedingly delicate, it will show indurations more distinctly than that of the adult.

The Care of the Skin.—Stretching or harsh manipulation of the skin of infants will tear or traumatize it. Irritating soaps should not be used. The drying of the skin should be carried out gently. The skin in the groin and axilla should not be unduly stretched lest rhagades or fissures result. In powdering the skin, a fresh pledget of absorbent cotton should be used as a powder puff, and all the excess of powder blown off, lest caking result. In some infants the wearing of flannel or wool next to the skin causes irritation and eruptions of different varieties. Such infants should wear a very fine cambric or linen garment next the skin, and over this the woollen shirt.

ECZEMA.

Eczema is a very common affection in infancy and childhood.

Some infants, otherwise in apparent health, suffer at times from a very mild eczema of the face, which appears chiefly on the cheeks, but which may also be present on the chin, forehead, and ears. The infants do not seem to suffer much, except that they scratch the eruption. The eruption is local. It may improve without treatment, but if there are conditions of traumatism and infection, it will grow worse. It is rarely moist, but, if scratched, it will bleed, and fissures or ulcers with bloody crusts will form.

Another form of eczema is pustular and vesicular. The skin of the face has a red, angry look. Here and there, patches of skin are covered with scabs; in other areas the skin is moistened by a serous or seropurulent exudate. This eczema is usually also present on the hands and arms. If the malady has existed any length of time, there is considerable thickening of the skin of the hands. The head and scalp may be affected.

Eczema is sometimes general. On the face, it is general and pustular; on the body, there are both the squamous and the pustular forms with all the various gradations between. There are crusts, rhagades, and areas of superficial loss of tissue.

The infants scratch and are uneasy and restless at night, but the general health is excellent and the appetite and digestion are good. The weight increases. If the eczema is general, the infants sometimes become puny. They scratch the eruption, constantly causing the surface to bleed. The body is sometimes one raw, suppurating surface. The lymph-nodes connected with the affected surface are enlarged. Such enlargements should be differentiated from those of pyogenic origin.

A very troublesome form of eczema is the impetiginous or pustular variety. The pustules burst and leave the surface covered with dried crusts of pus. This form may affect any part of the body. Of especial interest, and in a class apart, is the so-called impetigo faciei contagiosa. This is a contagious pustular eczema. It affects by predilection the upper lip and the alæ nasi. The pustules break down and leave dry crusts of a golden-yellow color. The anterior nares may be blocked up by these crusts. This variety of impetigo may in children spread over the whole surface and the extremities. I have seen it affect several children in a family. There can be very little doubt as to the infectious and contagious nature of the malady. Eichstedt, Lustgarten, and others have, with cocci obtained from the pustules, succeeded in inoculating the malady on the human subject.

Intertrigo (eczema intertrigo) or erythema intertrigo is one of the forms of erythema which develop by maceration into an eczema. Intertrigo is found in the folds of the neck, axilla, and groin, in well-nourished, rather obese infants. It is at first acute, but may become chronic. There is at first a slight redness of the folds of the skin (erythema). If through neglect the epidermis is allowed to macerate, excess of secretion results and the collected secretions decompose; the surfaces may become eroded, and ulcerations result. In some cases there are lineal ulcers in the groin. In others, the ulcers may become coated with a pseudomembrane. In rare cases actual necrosis of tissue results. Some anæmic infants present a tendency to rhagade formation, not only in the groin, but also around the anus and lips. The intertrigo may have the color of copper, instead of the bright-red hue of an ordinary eczema. In such cases there is always a possibility that the intertrigo may be of syphilitic origin. If there is no great panniculus of fat, and if with the intertrigo there appear erythema and fissures between the toes, and glossiness of the skin on the plantar surface of the feet, there are additional grounds for assuming that there is a syphilitic element. Intertrigo, like other skin eruptions, may be accompanied by enlargement of the lymph-nodes leading from the region affected. In obese infants, the umbilicus may also be the seat of eczema, which results from the accumulation and decomposition of secretions.

Seborrhœa capillitii is an eruption on the scalp of infants and children, which is classified by Unna as a form of eczema. The scalp is covered with a coating of yellow or discolored sebum, which consists of fat, desquamated epithelium, and hair. If allowed to accumulate, it is sometimes of considerable thickness and may be detached from the scalp. It then leaves a slightly reddened surface, which may bleed. In a short time the scalp may become glossy, and a new layer of the fatty secretion may form. This process may continue until the second or third year. This seborrhœic eczema has sometimes a cheesy odor.

Seborrhœa of the umbilicus has been mentioned. In infants and children there may also be seborrhœa of the prepuce. There are, in neglected cases, secretion and aphthous ulcerations of the folds between the glands and the prepuce and in the folds of the prepuce.

Of great interest to the physician is a form of intertrigo or eczema found on the buttocks and between the nates of infants. It occurs in infants who are not kept dry and whose urine decomposes easily if the diapers are not changed frequently. This is a most troublesome form of eczema. The nates are at first red, the skin then becomes glossy and brittle, and there may be extensive desquamation of the surface. This form of eczema or intertrigo may disappear under treatment, only to return if precautions as to cleanliness and dryness are not observed. Some of the children suffer from enuresis, and contract the affection through maceration of the skin by the decomposed urine, or from unclean diapers.

The **etiology** of eczema is still obscure. The conditions in infancy and childhood are favorable to the development of skin affections. The delicacy of the skin, its constant exposure to dirt and to irritants of all kinds, and changes of temperature, are etiologically important. All the children of a family may suffer from eczema. In such instances, there is a real hereditary tendency to the disease. The parents are sometimes similarly affected.

The influence of diet in causing eczema is not yet understood, but some authors are firmly convinced of the deleterious effects of certain articles of food. I have known urticaria to be caused by eating oatmeal and fruits, such as strawberries, and urticaria may be the beginning of eczema. In most cases eczema cannot be attributed to articles of diet. It is possible that in certain children the processes of metabolism are at fault. Though it has not been proved that all eczema is of an infectious character, there can be but little doubt that many forms are caused by the deleterious action of micro-organisms on the skin (Unna). In favor of this theory is the fact that in many parasitic skin affections eczema is an accompanying condition.

The **treatment** of eczema is exceedingly difficult. The external causes of irritation should be immediately removed. Attention to cleanliness is alone sometimes sufficient to cure an eczema. If woollen clothing is irritating to the skin, a substitute should be found and cotton or cambric should be worn underneath the wool.

The diet should be regulated. This is not an easy task, since it is not known what articles of diet produce eczema. If the infant is at the breast, the diet of the wet-nurse and her daily habits should be regulated. Even when the nurse takes simple food, and the milk is flawless, the infant may suffer from eczema. If the nurse is addicted to the use of beer, or vegetables, such as asparagus, the quality of the milk may be affected. The diet of a wet-nurse should not be changed more often than is necessary, else the secretion of milk may cease. If the wet-nurse has a rheumatic or gouty tendency, it is wise to change nurses. On the other hand, an infant may be overfed and excessively fat. In that case the intervals between nursing should be lengthened. To attempt to change the percentage of fat in the milk is not only of questionable utility, but is not always feasible. If the nurse is constipated, the bowels should be regulated, and she should take abundant exercise. Artifically fed infants are still more difficult to manage. If the infant is thriving, interference with the food percentage is not always clearly indicated. Artificially fed infants may also be overfed or the percentage of fat or proteids may be too high. There may, however, be eczema even when the composition of milk is proper for the infant, age and weight being taken into consideration.

If there are acidity of the stomach, excessive flatus, constipation, or green stools, regulation of diet is of more practical utility. In such cases it may cause the eczema to diminish. If there is stomach acidity, an alkali (lime-water) should be added to the food. Constipation and flatulence should be remedied. If the infant passes urine loaded with urates to such an extent as to cause a red deposit on the diaper, small doses of bicarbonate of sodium should be administered and lime-water should be mixed with the food.

Changes of diet are helpful only in those forms of eczema which are either general or disseminated over different parts of the surface. Seborrhœa and intertrigo are purely local affections, and are not influenced by changes of diet.

Local treatment is chiefly relied upon to improve the condition of the skin. In the acute or subacute forms soothing applications are utilized. The chronic forms are irritated into a state of reaction, and then treated like acute eczema. The treatment of acute local eruptions, such as intertrigo, consists first in keeping the parts scrupulously clean. After the bath the folds of the skin are mopped, dried carefully, and powdered, the excess of powder being blown off. This alone is sometimes sufficient to cure a slight intertrigo. Dusting-powders which contain carbolized preparations irritate the skin. A good powder has the following composition:

℞ Zinci oxidi	ʒiv (16.0).
Amylum	℥ij (60.0).—M.

Equal parts of zinc and starch powder make an equally good powder. These ingredients should be ground to an impalpable powder. In the severer forms of intertrigo, the parts should first be anointed with ointment having the following composition:

℞ Resorcin	gr. ij–iv (0.12–0.24).
Adeps benzoinati	℥j (30.0).

M.—The lard should be washed.

The ointment should be removed from the folds of the skin with a pledget of lint. The skin after being thus left in a slightly greasy state is powdered, the excess of powder being blown off. If there are lineal ulcers in the groin, they should be lightly touched once a day with a 2 per cent. solution of nitrate of silver, to promote granulation. The ointment should then be applied with a small piece of lint.

In squamous eczema, which is a red or pustular eczema of the face, scalp, and hands, the first question that arises is whether the patients should be bathed. An infant should be kept clean, and there is only one satisfactory method, and that is the bath. If there is eczema of any part of the surface, the bath water may be liberally impregnated with bran. A gauze bag filled with a measure of bran is put into the bath and the bag squeezed until the water becomes turbid. If a minute quantity of bicarbonate of sodium is added to a bath prepared in this way, the effect on general eczema is decidedly soothing. The skin is gently dried after the bath and powdered. If the whole trunk is involved, it is best that the parts of the surface should be treated in succession. The face or an arm is covered with an ointment applied by means of a piece of lint, or the ointment is simply rubbed on the skin after the bath. It is not feasible to wrap the whole body in lint and ointment; with certain drugs, such as resorcin, absorption would occur. The ointments should be applied after the crusts and pustular accumulations have been removed. All ointments should be made up with washed benzoinated lard. Vaseline is very irritating to some forms of eczema. Of the emollient and soothing ointments, diachylon, zinc, and bismuth hold a leading place. A very good ointment for general use in rhagades and squamous eczema is the following, which is one of Kaposi's formulæ:

℞ Resin. benzœa pulv.	ʒj (4.0).
Axung. porc.	℥v (150.0).
Digere cola adde.	
Zinc. oxidat.	℥j (30.0).
M. et. ft. unguentum.	

If made up properly, this is an excellent cosmetic ointment for use in dry eczema. If the skin is dry and thickened, a 1 per cent. β-napthol applied twice daily will soften it. If this treatment proves irritating, a zinc ointment may be applied immediately afterward.

In many cases of chronic eczema Lassar's paste is beneficial:

℞ Acidi salicyl.	gr. xxx (2.0).
Zinci oxidat. Amylum	āā ℥j (30.0).
Vaselin.	℥jss (45.0).
M. et ft. paste.	

The following ointment is also excellent:

℞ Acidi salicylici	gr. xv (1.0).
Ung. zinci oxidi	℥ij (60.0).—M.

The tar salves and mixtures are useful in cases of chronic eczema in which there is little or no moisture.

℞ Ol. rusci	ʒj (4.0).
Ungt. zinci ox.	℥j (30.0).
M.—For external use.	

or

℞ Ol. fagi	ʒijss (10.0).
Glycerin.	ʒj (4.0).
Ung. diachylon	℥jss (45.0).
Balsam. Peru	♏ xxx (2.0).—M.

In cases of red eczema of the face, the ointment is best applied on a mask made of lint.

In that form of intertrigo which results from the irritation of urine, the condition of the diaper is frequently the chief source of trouble. It is often damp or too thin. As a result, whenever the infant passes urine, the diaper becomes saturated with it and decomposition takes place. A piece of absorbent gauze as large as the diaper should be placed next the skin, and renewed whenever it becomes moistened. The skin is dried and the ointment applied on the gauze. Intertrigo is quickly cured by this treatment.

Treatment of Seborrhœa of the Scalp.

The accumulated sebum is moistened with oil, or a piece of lint moistened with olive oil or any indifferent oil is applied at night. In the morning the crust of sebum will have softened sufficiently to allow of its removal with green soap and water. After the parts are well cleaned, a salicylated ointment, 0.5 to 1 per cent., is applied daily. The ointment should be sparingly applied in order that it may not irritate the parts. Seborrhœa should be treated for some time after it is apparently cured, or it will return. In older children who have abundant hair, the seborrhœa accumulates at the roots and the scalp has an odor. The head should be thoroughly shampooed once a week; after the shampoo, an exceedingly small quantity of cosmetic hair oil should be applied to the scalp once a day.

ERYTHEMA MULTIFORME.

(*Erythema Nodosum; Erythema Exudativum.*)

Erythema exudativum is divided into two forms. The acute form includes erythema multiforme and nodosum, and is an acute infectious disease (Lewin). The exudative form occurs frequently in infants and children. Of 40 of my cases, 10 were under two years of age. The form of erythema known as erythema nodosum begins with general malaise and sometimes with fever, which may be quite high. There is pain in the joints and over the areas affected. These areas are raised and are purple or bluish; the skin is tense and the parts affected are very painful. The nodes vary in size. They first appear chiefly on the extensor surface of the tibiæ. The extremity sometimes looks as if it had been beaten. This form of erythema is perhaps allied to hemorrhagic diseases, such as peliosis. In a case of peliosis rheumatica which I saw recently there were erythematous and painful nodules on the hands. Antitoxin may cause exudative erythema. As is well known, such toxic infection also involves the joints. The symptoms are fever, pain in the joints, and extensive erythema nodosum. I have seen such a case in a subject, who had received an immunizing injection. Within six hours, the legs, knees, and thighs were swollen and the seat of this peculiar erythema.

French writers speak of the frequency of cardiac disease in erythema nodosum, and of its relationship to rheumatism. I have carefully studied 40 cases for signs of cardiac disease, and could find only 3 cases with systolic murmur at the apex. I have recently seen 2 others. In my opinion, true endocarditis is not a very common complication of erythema nodosum. In only one case did the murmurs appear to be serious. The disease lasts only a few days, but there may be relapses.

The second form of chronic erythema resembles the acute form. The nodules are flat and deep, and are not raised much above the surface. They appear chiefly on the lower extremities of badly nourished children. They are less painful than in the acute form. After a time they disappear leaving no sign of their presence.

Treatment.—Cases of erythema of the acute form are treated with sodium salicylate and a diet of milk at first, fruit-juices and beef-juice being given later, and local applications of oil of wintergreen to the painful areas.

FURUNCULOSIS.

(*Folliculitis Abscedens or Perifolliculitis Abscedens.*—ESCHERICH.)

This affection of the skin is very common in infancy and childhood, and occurs chiefly in badly nourished, marantic babies, who suffer from gastro-enteric and pulmonary infections. The disease is due to an invasion of the deeper layers of the skin by staphylococci. These have been found in the pus and in the sweat and sebaceous glands of the skin (Escherich). In the mild forms of furunculosis there are one, two, or more furuncles on the forehead, scalp, occiput, and neck. Sometimes the furuncles are large and the skin is riddled with them, but as a rule they do not communicate with one another. In aggravated cases, furuncular abscesses occur on the trunk and on the upper and lower extremities. When the furuncles or boils become very numerous, they play a leading rôle. Many children in institutions succumb to this affection. The condition closely resembles a form of sepsis.

The **treatment** of these cases is simple. I have administered alkalies, such as bicarbonate of sodium, internally. The effect on the general process is excellent. I have also given sulphide of calcium in grain ½ doses (0.03) with good effect. The infant is bathed in bran daily. Too many of the abscesses should not be opened at once, and they should not be opened until they point and the skin over them becomes reddened. If they are opened earlier, the results are not so good. After the abscesses are opened, the pus is expressed and a moist dressing applied. The abscesses heal easily. As in other septic affections, the patients should be stimulated and carefully fed. Small furuncles appearing only on the face need not be opened. The application of a 2 per cent. salicylated ointment twice daily softens the pustules and causes the contents to be discharged.

SUDAMINA.

(*Miliaria Alba; Miliaria Rubra.*)

Sudamina is an affection occurring in infants and children during very warm weather. In the form called miliaria alba the epidermis at the openings of the sweat-glands is raised by a minute serous exudate and small vesicles are formed. There is no inflammation of the skin. In a second form, the same process takes place, with the presence of a minute focus of inflammation and redness at the opening of the glands. Some of the vesicles are pustular. There are also numerous papules of eczema. There is a slight infection of the skin about the opening of the sweat-glands. Both these conditions are irritating, but in no way serious. The skin should be kept scrupulously clean and dried with powder. Woollen fabrics should not be worn next the skin. If the condition becomes severe, bran baths and a bland zinc or diachylon ointment should be used. Sudamina of both varieties are met with in scarlet fever dermatitis.

DERMATITIS EXFOLIATIVA.

(RITTER VON RITTERSHAIN.)

This affection is peculiar to the newborn infant. Ritter in 1878 described an epidemic. In 1895, Escherich published an account of a small local outbreak in Gratz.

Nature and Etiology.—It was first suspected by Ritter to be one of the septic infections of the newly born infant. His view has lately been supported by Escherich.

Occurrence and Symptoms.—The disease appears from a few days to two weeks after birth. It usually occurs in poorly nourished infants, but may affect apparently healthy infants of normal weight. Boys are more frequently affected than girls. The affection is preceded by the appearance of a diffusely red erythematous or dark swelling of the general surface. The skin is thickened, soft, macerated, and velvety to the touch. The epidermis can be moved on the corium beneath. The pressure of the clothing or bedclothes may also produce this effect. Minute vesicles appear, and coalesce to form larger vesicles or bullæ. Vesicles or bullæ of large size which may be either partly filled with serum or empty are formed. They are never tense, and finally open or tear, leaving the red moist corium exposed. The surface of the body has a beefy-red color, and is covered here and there with patches of dry, adherent epidermis; in other areas, the corium is exposed. There are rhagades at the angles of the mouth and on the trunk. The upper extremities become affected later than the lower ones. Whole areas of the trunk and body are denuded of epidermis. After the vesicles burst and leave the corium exposed, the epidermal layer of the skin is still adherent in places, while the desquamated skin is rolled up into cord-like masses and hangs loosely exposed. If recovery takes place, the corium becomes covered with a delicate epidermis, which gradually assumes the normal pinkish-white hue. Some cases may run an afebrile, others, a febrile course.

Course and Prognosis.—A few of the cases recover. Ritter lost 50 per cent. of his cases, and Escherich 90 per cent. The infants may die from the sixth to the tenth day or after the third week, when much of the skin has undergone retrograde changes. The cases may show umbilical infection or lobular pneumonia pointing to the septic nature of the disease.

Treatment.—The infants are kept warm by artificial means, such as warming bottles or an incubator. They are not bathed. The skin is protected by the application of bland salves or gauze moistened with a mixture of linseed oil and lime-water (Escherich). Some physicians add a small quantity of salicylic acid to the salves. As soon as the skin has become dry, Lassar's paste and powdered zinc are applied.

CONGENITAL ICHTHYOSIS.

(*Cutis Sebacea.*)

Ballantyne gives an exhaustive description of this affection, which is really a perpetuation of a fœtal condition into post-natal life. The fœtal skin has a tendency to seborrhœa. This is apparent after birth, and is evident during infancy as seborrhœa of the scalp. The seborrhœa may affect different parts of the body and may form thin shining scales on the surface of the skin. There may be secondary eczema. The mild forms may, with ordinary cleanliness and the application of bland salves, disappear a few weeks after birth. The form described by Hebra and Kaposi as ichthyosis congenita is an extreme example of the tendency of the fœtal skin to the formation of sebum or vernix. The increased secretion continues after birth. The infant appears to be covered with a horny mass which almost envelops it. This parchment-like covering is absent at the mouth, eyes, anus, and on the scalp. The surface is firm and of a yellow or brownish-red tint (Escherich). The hardness and brittleness of the skin render motion painful. The infant is enclosed as if in case-armor. The face has a mask-like expression. The skin is broken in places, especially at the joints. At these fissures the true skin is seen. At the broken spots, the sebum is seen to be composed of lamellæ, from the posterior aspect of which project warty excrescences corresponding to the lanugo and openings of the sebaceous glands. These may be removed from the skin. If the infant lives, the layers of sebum are thrown off gradually, and the skin is left with a general seborrhœa of the ordinary type. Escherich predicts a favorable course in most of these cases, but some die shortly after birth. Pathologically there is a great thickening of the rete Malpighii; the corium shows no changes; the sebaceous glands are atrophied or the seat of fatty degeneration; the sudoriparous glands are normal. After the layers of horny sebum have peeled off, the skin underneath appears pink or red or shining, and is covered with seborrhœal scales.

The **treatment** consists in the application of emollients and in washing the skin daily or bathing the infant in permanganate of potassium (grains xv (1.0) to the bath water). Salicylic and boric ointments are applied after the baths.

PEMPHIGUS NEONATORUM.

Pemphigus neonatorum is a contagious infectious disease of the skin occurring in the newborn infant. It has also been observed later in infancy. It usually appears at the end of the first or second week, and affects the whole surface, except the palms of the hands and the soles of the feet. There appear on the surface of the trunk and extremities small and large vesicles containing cloudy serum. These burst and leave a round patch of skin, which dries and is covered with yellowish scales. The vesicles may attain the size of bullæ. They may be discrete or involve the whole body, so that the surface is after a time denuded of the epithelial layer. The disease may in the beginning be confounded with dermatitis exfoliativa. The vesicles may appear in crops; the recurrences may extend over a period of from two to four weeks.

There are two forms, in one of which the disease is mild; in the other, it runs a malignant course, and from the outset large areas of skin are denuded of epithelium by the bursting of enormous bullæ. The infants pass into an asthenic condition, refuse nourishment, and die in a few days. Both forms appear in epidemics. The disease occurs sporadically. The essential cause is still obscure. Strelitz, Demme, Almquist, and Escherich have isolated a white staphylococcus from the serum of the vesicles. Its rôle as an etiological factor is not as yet understood. Escherich is inclined to class this form of pemphigus with other infectious skin diseases, such as the impetigo of Wilson or Bockhart, and folliculitis abscedens, in which certain conditions favor serous infiltration of the horny layer of the skin and extensive desquamation from the corium. He believes the exciting cause to be the pus cocci found in other forms of impetigo. Escherich has suggested the use of the name "Impetigo Bullosa Neonatorum or Infantum" for this affection.

The **prognosis** is favorable if the process confines itself to the superficial layers of the skin. If the deeper layers are attacked, abscesses and general sepsis result.

Treatment.—Escherich recommends that the affected parts be washed with soap and water, and dressed with a 2 per cent. ointment of white precipitate. Baths are not given. Those who are interested in the epidemiological aspect of this disease will find the monograph of Richter exhaustive.

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